

## **Supporting Information**

### **Supplemental References**

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## **Supplemental Experimental Procedures**

### **Tasks**

In order to investigate the mechanisms underlying working memory, we trained three monkeys on three different working memory tasks. Task one (Fig 1A) required memory for a sample object over a delay (which was variable at 0.5-1.2s for 20 recordings and fixed at 1.2s for 9 recordings). The other two tasks engaged working memory for a spatial location; however, they differed in the presentation of a masking stimulus during the delay. In task two (Fig 1B), after a location was cued by a red dot, the animal had to maintain information as a visual mask (red dots at all possible cued locations) was presented through a variable delay (2.2 to 2.7s). In the third task, the monkey had to similarly maintain one of four possible cue locations after a short sample epoch (0.295s); however, during the subsequent 1s delay, there was no visual mask, only the fixation point was shown during the delay. The idea in comparing these tasks was to show that independent of both the content of the working memory and the presence or absence of distractors during the delay, the neural patterns were comparable. Those phenomena we observed, therefore, were general hallmarks of working memory, and for the purposes of this paper all of the data was pooled together (except where indicated). Behavioral performance was high for each of the tasks/monkeys (masked delayed saccade/monkey C: 85%, visual search/monkey S: 77%, delayed saccade/monkey P: 99%). Only correct trials are included in the present analysis, except for Fig. S11, which is an analysis of the error trials pooled over the masked delayed saccade and visual search tasks (there were insufficient error trials from the delayed saccade task).

## Recordings

We acutely inserted between 1 and 3 laminar probes into cortex in every recording session. Our recordings included a large portion of the macaque frontal cortex, spanning dorsal premotor regions to lateral prefrontal cortex. In Fig. 1G, the colored dots represent recording sites from each of the monkeys and their respective frontal areas. In addition to the areas that are depicted, we also performed several recording sessions from deeper midline structures, such as the supplementary motor area (SMA) and the anterior cingulate cortex (ACC).

We identified cortical areas based on location relative to anatomical landmarks and used area labels in Saleem and Logothetis (1). MRIs were taken of each individual monkey with the recording grid in place, filled with water, which created a marker to co-register each possible electrode trajectory with the animal's anatomy. Area 8A was defined as recordings on the gyrus posterior to the principal sulcus, and anterior to the arcuate sulcus. This area corresponds to area 8Ad/v in (1). Dorsal Lateral Prefrontal Cortex (DLPFC) were recordings dorsal to the principal sulcus, anterior to Area 8A, and ventral to the upper branch of the arcuate sulcus. This area corresponds to area 8Ad in (1). Ventral Lateral Prefrontal Cortex (VLPFC) were recordings ventral to the principal sulcus, anterior to Area 8A, and dorsal to the lower branch of the arcuate sulcus. This corresponds to area 8Av and 45A in (1). Area Dorsal Premotor (PMd) was defined as the area on the gyrus anterior to the spur of the arcuate sulcus, posterior to the rostral end of the arcuate sulcus, and dorsal to the arcuate sulcus. This area corresponds to cytoarchitectonically defined area F2 and F7 in (1), and is contains part of area SEF

(Supplementary Eye Fields). The deeper midline structures, folded immediately underneath PMd, encompassed the SMA (Supplementary Motor Area) and ACC (Anterior Cingulate Cortex). The ACC corresponds to area 24c in (1). The SMA corresponds to area F3 in (1). Recordings in ACC/SMA were performed in the same trajectories as those for the PMd recordings, but electrodes were advanced deeper (see Fig. S12). Area 8B recordings were performed anterior to the end of the upper branch of the arcuate sulcus and corresponds to area 8Bd in (1). Details on probe spacing, site number, sampling coverage, and areas sampled per monkey/task, are provided in Supplemental Table 1.

We used linear laminar probes (“U probes” and “V probes”) from Plexon (Dallas, TX) with a variety of inter-site spacing (100, 150, or 200um) and contact numbers (16, 24, or 32 contacts per probes), giving a sampling range between 1500 and 4500um (see Supplemental Table 1 for details). Probe geometry (inter-site spacing, channel count or U/V type) had no qualitative impact on the data we report here. Because the contact spacing ranged between 100 to 200um, we used cubic spline interpolation to up-sample the data and organize it into the same depth coordinates. To do so, we up-sampled to 100um spacing (if the spacing was already 100um, no interpolation was performed). Each individual probe was considered a unit of observation for our analyses in Figs. 2-5. For the analysis of thresholded units (Figs. S4-7), the unit of observation was thresholded spikes.

In Fig. 1C-E, MRIs are plotted with sample trajectories superimposed (red lines) in each of the monkeys. These trajectories were approximately perpendicular to the cortical sheet, allowing a relatively unbiased sampling of all cortical layers. All of the data was recorded through Blackrock headstages (Blackrock Cereplex M, Salt Lake City, UT), sampled at 30 kHz, band-passed between 0.3 Hz and 7.5 kHz (1<sup>st</sup> order Butterworth high-pass and 3<sup>rd</sup> order Butterworth low-pass), and digitized at a 16-bit, 250 nV/bit. All LFPs were recorded with a low-pass 250 Hz Butterworth filter, sampled at 1 kHz, and AC-coupled. In monkey C, the reference was the headpost, in monkey S, the reference was internal to the metal shaft surrounding the probe itself. In monkey P the reference was a nearby guide tube sitting on the dura. Some U/V probes had noisy channels (average power greater than 2 standard deviations above the mean of all channels, this occurred on less than 5% of all channels), which were removed prior to analysis.

## **Lowering Procedure**

In order to place the contacts of the laminar electrode uniformly through the cortex, spanning from cerebrospinal fluid through the gray matter to the white matter, we used a number of physiologic indicators to guide our electrode placement. First, the presence of a slow 1-2 Hz signal, a heartbeat artifact, was often found as we pierced the pia mater and just as we entered the gray matter. Second, as the first contacts of the electrode entered the gray matter, the magnitude of the local field potential increased, and single units and/or neural hash became apparent, both audibly and visually with online spike thresholding. Once the tip of the electrode transitioned into the gray matter, electrodes were lowered slowly with minimal vibration an additional 1-2mm. At this

point, the electrodes were allowed to settle for about 5 minutes, and then we began a visually evoked potential paradigm.

During this paradigm, we flashed a white screen on for 50ms with 500ms pauses while the animal's eye position was detected on the monitor. We repeated this about 200 times, and then calculated a power profile and Current Source Density (CSD) over the contacts of each of the implanted laminar probes. To compute the CSD, we cut the data into trials at the flash onset, obtained the evoked response to the flash across trials, and computed the evoked responses' second spatial derivative (2). The CSD reflects regions where ionic currents are flowing into neurons (caused by a net depolarization of surrounding neurons), and an early sink (where the CSD goes negative within 100ms of stimulus presentation) in response to visual input has been mapped to layer 4 in visual and auditory cortex (3, 4, 16). Layer 4 in these regions receives the bulk of thalamic or bottom-up sensory inputs. In frontal cortex for areas that are agranular (area PMd), an early sink corresponds to the bottom of layer 3 (17), which receives visual sensory inputs (5). In the case of granular prefrontal cortex (containing a well-defined layer 4: areas DLPFC, VLPFC, and 8A/B) we expect the early sink to correspond to layer 4, which receives thalamic input from mediodorsal nucleus of the thalamus as well as sensory cortical areas (18, 5).

We sought to position this current sink at approximately the middle contact of the probe. In addition to using the CSD for electrode alignment, which was more or less noisy, we also examined whether power in the alpha/beta (10-30 Hz), and gamma bands (30-80 Hz) varied across the probe. This was used to determine whether the probe had entered cortex, which was accompanied by a dramatic increase in LFP power across frequencies. The power variance also confirmed whether we had placed the probe in a cortical gyrus (recordings in cortical sulci had minimal power variance), power variance over contact was minimal. We visualized these power estimates at each contact and normalized the power estimates by the maximal power within both of these bands. We found that the crossings of the normalized powers within the alpha/beta and gamma bands nearly always occurred within one or two sites of the earliest sink. Since these power profiles correlated strongly with the CSD results, we used these power profiles to also guide our decisions on whether or not we lowered the electrode any further. Finally, we were also careful not to penetrate by more than an electrode contact or two into the underlying white matter. The white matter was characterized by the predominance of upward going spikes, and the absence of any CSD (i.e. there was markedly low LFP variability). Thus, all of these physiological markers (heart beat artifact, CSD to screen flashes, increase in power at all frequencies upon leaving CSF and entering cortex, power variance in the alpha/beta and gamma bands, presence of spikes/MUA hash, and white matter signatures of lower LFP variance and upward going spikes) were used to determine probe placement. Once the probe was fully lowered, we allowed it to settle for an hour, and then began a longer flashing sequence of 800-1000 trials for offline CSD analysis.

Importantly, the laminar alignment, e.g., each session's "zero point" (corresponding to layer 4/bottom of layer 3), was determined entirely on the basis of offline analysis. It was

based on the site at which current sinks were detected within a time window of 40-180ms of flash onset. In monkey P, due to noise during pre-task flash trials, CSD sinks for alignment purposes were calculated based on the same time window, but in response to sample onset. Across sessions, aligning depth zero to the first significant CSD sink in response to sample onset or the pre-task flash trials gave essentially identical results: the relative gamma and alpha/beta power profiles with respect to the initial sink (Fig. S1) had a Spearman correlation of 1. CSDs were calculated for each trial, taking a spatial integration of between 350-400  $\mu\text{m}$ , to be as consistent as possible given our probe geometries (i.e., using 100 $\mu\text{m}$  inter-electrode spacing, we integrated every 4<sup>th</sup> contact, for 200 $\mu\text{m}$  spacing, every 2<sup>nd</sup> contact, and for 150 $\mu\text{m}$ , every 3<sup>rd</sup> contact). We subtracted the CSD at the pre-flash baseline (50ms to flash onset), and then z-scored the CSD data over trials by dividing the raw CSD values by their standard error. We then assessed which CSD contact first achieved a z-score of less than -4 (negative CSD values correspond to current sinks), and which lasted at such a level for at least 6 ms. We assigned this contact as the first significant sink, and this provided the zero (the middle layer) for each penetration.

Relative to this zero point, the average distance to the CSF outside of gray matter was 0.9mm, estimated based on the total power of the LFP (when power across all frequencies decreased below 30% of the contact with maximal power, similar to (17)). We also measured the cortical thickness for each of the 60 penetrations based on each individual monkey's MRI. To visualize the specific electrode tracks, we obtained an MRI of each animal with their respective recording chamber and recording grid in place. Filled with water, the grid lumens could be tracked, and electrode trajectories projected onto the cortex and its folds (Fig. 1D-F, Fig. S12). The mean cortical thickness across the penetrations in this study was 2.4mm (measured manually using each animal's MRI, the corresponding recording trajectory, co-registered, visualized, and measured using Osirix software, Geneva, Switzerland). Thus to span the full average cortical distance in our analyses, laminar profiles were plotted from 0.9mm above the sink (the corresponding depth of the average gray matter / CSF transition) to 1.5mm below the sink (average gray matter / white matter transition). Because our recordings span a number of frontal areas that are agranular (containing no discernable layer 4, PMd), dysgranular (containing only a very thin layer 4: SMA and ACC) and granular (containing a well-defined layer 4: 8A, 8B, DLPFC, VLPFC), we chose not to classify particular contacts to specific layers, as the laminar widths and organization could be slightly different over areas. Instead, we chose a more general classification system that could be applied to all areas, grouping contacts at and above the sink as superficial layers (corresponding to layers 1-3) and contacts below the sink as deep (corresponding to layers 5-6). The distance from the sink was a proxy for laminar position.

## **Analysis**

All analysis were performed with customized MATLAB scripts and with Fieldtrip software (7). Given probe movements across a day, we smoothed across contacts on all analyses. This smoothing was symmetric. For the analyses based on LFP and

analog MUA (Fig. 2-5, Figs. S1-3, S8-12), which could be defined for all contacts, we smoothed 200um above and below the contact of interest. This smoothing parameter equaled the distance between the contacts of the coarsest probe used in this study. For the analysis based on thresholded MUA (Figs. S5-7), due to the much sparser nature of the signal, we increased the smoothing to go 500um above and below the contact of interest.

For Fig. 2, power was calculated based on 700 ms segments of data (200 ms prior visual stimulation to 500 ms post visual stimulation), which were tapered with Hanning windows. After these steps, we applied a fast Fourier transform.

For Fig. 3A, we present the MUA change from baseline from 0.150 s post sample offset to 1 second post sample offset. The delay period in this task (search), varied between 0.5 to 1.2 seconds. We determined the average for each time point in the delay based only on trials in which the delay lasted at least until that time point.

To compute phase amplitude coupling (also known as cross-frequency coupling, used in Fig. 5 and S9-10) during the delay or baseline periods we applied the Hilbert transform on two sets of band-passed filtered data: one, band-passed for lower frequencies (4-22 Hz), and the other band-passed at higher frequencies (50-250 Hz). The modulation index (19) was used to quantify the extent to which the faster frequency power deviated from a flat distribution over different phases (taking 18 non-overlapping equally spaced phase bins). We took the phase of the lower frequency Hilbert transform, and the amplitude of the higher frequency Hilbert transform. When no phase-amplitude coupling exists, average gamma amplitude over the 18 non-overlapping alpha/beta phase bins will be similar. This will result in a low entropy value of gamma amplitude over the phase bins. In contrast, when phase-amplitude coupling exists, gamma amplitude will be more concentrated at some phase bins compared to others, resulting in greater entropy over phase. The modulation index is a measure of the degree of entropy (non-uniformity) that is present in this distribution.

The computation of Granger causality in the frequency domain requires the estimation of two quantities: the spectral transfer matrix ( $H(\omega)$ ), which is frequency dependent, and the covariance of the model's residuals ( $\Sigma$ ). The spectral transfer matrix defines how power in one channel is transferred to other channels, at each temporal lag. The model's residuals is not a function of frequency, and defines the amount of variance that is left unexplained by the linear model,  $H(\omega)$ . Traditionally,  $H(\omega)$  is computed in a parametric (model-based) fashion by first fitting an autoregressive model to the data, and then Fourier transforming the model (8). However, it is also possible to compute,  $H(\omega)$ , and thus Granger causality, directly from the spectral transform of the data. In brief, the following fundamental identity holds:  $H(\omega)\Sigma H(\omega)^* = S(\omega)$ , with  $S(\omega)$  being the cross-spectral density matrix at frequency  $\omega$ . Starting from the cross-spectral density matrix ( $S(\omega)$ ) it is possible to factorize the cross-spectral density matrix into a noise covariance matrix ( $\Sigma$ ) and spectral transfer matrix ( $H(\omega)$ ) by applying spectral matrix factorization (9) —which provides the necessary ingredients for calculating Granger causality. The nonparametric estimation of GC has certain advantages over parametric

approaches in that it does not require the specification of a particular autoregressive model order.

Power and cross-frequency coupling analysis were calculated on unipolar data. Granger causality (GC) was computed through a nonparametric spectral matrix factorization of the Fourier transforms of bipolar data (8) during the delay (Fig. 5A). Bipolar derivation is a recommended pre-step prior to Granger causality analysis, as the presence of a common reference can lead to spurious results (10, 11). However, when bipolar derivations are too close to one another and cortical sources are synchronous, the effect of bipolar derivations is to remove the oscillation of interest. Therefore, bipolar derivations were performed between adjacent probes, always taking the contacts that were aligned to the same depth (relative to the early significant CSD sink at each probe). This was only possible when two probes had been simultaneously lowered to the same cortical area (and were between 2-4 mm apart), which occurred a total of 10 times (6 in area PMd in the masked delayed saccade task, 4 in VLPFC in the search task). To control for possible contributions of signal-to-noise differences in driving GC, we performed time-reversed Granger testing (12). In all cases, time-reversing the signals either reduced the dominant directionality or flipped it (from ascending to descending), confirming that signal-to-noise differences could not explain the ascending dominance of GC.

For the analysis of gamma and alpha/beta bursts, we used wavelets to capture deviations in power over time that could change quickly in both time and frequency, reflecting the non-stationarities of interest. Power was computed at equally-spaced frequency bins between 4 and 150 Hz. For each frequency, we used wavelets with a width of 5 cycles, and estimated power every 5ms. Bursts were detected as epochs when power exceeded a threshold of mean +2 standard deviations for at least three cycles, given the center frequency of that burst. Bursts in the gamma-band were detected between 50 to 150 Hz, reflecting the fact that previous findings have shown this to be the upper bound for oscillatory gamma responses (20). Bursts in the alpha/beta range were detected between 4-22 Hz, chosen according to frequencies which had peak power in the deep layers. To minimize the contribution of spikes to the gamma bursts, we removed bursts that contained significant increases in power across all gamma frequencies (50-150Hz), reflecting the fact that spikes are expected to contribute power to this entire band (13) and that bursts should contain power in a relatively narrow frequency range. To calculate whether the delay-period bursting rate contained WM information, we summed the number of bursts around a sliding window (200ms for gamma and 400ms for alpha/beta), and assessed whether the burst rate reliably distinguished between the different cues using an unbiased measure of information, percent explained variance (omega-squared; (14)). Information was then averaged across the delay period.

For the power-power correlation analysis, we correlated power between alpha/beta and gamma in the delay epoch for all possible combinations of alpha/beta providing- and gamma providing-channels, on a trial-by-trial basis (Fig. 5E). We performed this correlation on sessions where the delay length was fixed (total N=13, 4 sessions from



the delayed saccade task and 9 sessions from the visual search task) and monkeys could predict the timing of the test stimulus/go cue. Previous work has shown that predictability of the delay leads to a strengthening of gamma and weakening of alpha/beta (20).

### **Analysis, MUA Activity**

For the analysis of the analog multi-unit activity (MUA, Fig. 3, S3 and S8) we band-pass filtered the raw, unfiltered, 30kHz sampled data into a wide band between 500-5,000Hz, the power range dominated by spikes. The signal was then low-pass filtered at 250Hz and re-sampled to 1,000 kHz. The advantage of using this signal is that every channel can now be used to estimate a MUA signal, even channels which do not contain isolatable spikes.

### **Analysis, thresholded spikes**

For the analysis of thresholded spikes (Figs. S4-7), we began with the raw, unfiltered, 30 kHz sampled data. We re-referenced each contact's signal to the global mean across all contacts, applied a 6<sup>th</sup> order 250Hz, high-pass Butterworth filter, and then z-scored each signal by its own mean and standard deviation. We next identified spiking by identifying those time periods when the z-scored signal fell below 5 standard deviations of the mean noise floor. In order to ensure that all of these spikes were appropriately captured in time, we further extracted 10 samples before and 24 samples after threshold crossing from the original non-thresholded, 30 kHz signal. This new data was up-sampled by 2 using cubic splines, and the global minimum of each of these snippets was identified. If any spike was counted twice, because of more than a single threshold crossing within this time interval, they were rejected. Finally, to rule out spurious thresholds due to random noise or unit drift, we included units for further analysis if they maintained a firing rate of at least 2 Hz for at least 25 trials. The entire spike analysis pipeline was fully automated.

### **Analysis, statistics**

To assess whether different laminar data features (delay-period MUA and gamma/alpha-beta information, deep-to-superficial CFC vs. other laminar combinations of CFC, ascending vs. descending Granger causality, power-power correlations in different laminar compartments) were significantly different in superficial vs. deep layers, we performed sign tests over laminar probes. The advantage of using a sign test was that the test had minimal assumptions about the underlying distributions. For these different metrics (for example, delay MUA in superficial vs. deep layers in Figure 3H), we first averaged across all superficial and all deep contacts on each session. If superficial MUA was greater than deep, we converted this to 1, otherwise we converted the value to -1. The input to the sign test was the result of this superficial vs. deep comparison (a vector of 1's and -1's). The number of data points was 60 (total number of laminar probes in the study) for comparing relative power in superficial vs. deep

layers (Fig. 2A), delay period MUA in superficial vs. deep layers (Fig. 3H), for comparing delay vs. baseline bursting and information in superficial vs. deep gamma bursts (Fig. 4), for comparing CFC between the 4 different laminar compartments (5C), and for the delay vs. baseline CFC comparison (Fig. 5D).

Power correlation analysis (Fig. 5E) used 13 data sets due to the requirement of a fixed delay-period. Also, Granger causality analysis (Fig. 5A) used 10 data sets, due to the requirement of bipolar re-referencing across probes. Here, the paired sets of data that were compared (and transformed to 1's and -1's) were average GC in the superficial to deep direction vs. average GC in the deep-to-superficial direction.

To compute correlations between laminar profiles, we first computed the average profile for a particular data feature (relative power, cross-frequency coupling, or delay-period MUA modulation), taking the average across either all laminar profiles, all laminar profiles within a task, or all laminar profiles within an area (as indicated in the text). We then computed Spearman's rank correlation (Rho) to assess, for example, the hypothesis that the grand-average gamma and alpha/beta power profiles (Fig. 2B) were correlated to the grand-average delay period MUA profile (Fig. 3G). The reason we used Spearman rank correlation is because the underlying distribution of the data (power/CFC/MUA profiles) was not normally distributed. The magnitude of Spearman's Rho is computed by calculating the Pearson correlation coefficient after rank ordering the two sets of variables. The p-value of this correlation is determined using a non-parametric test that does not make any assumptions about the underlying distribution. The non-parametric test proceeds as follows: a reference distribution is approximated by randomly permuting the laminar order of the data. This is equivalent to destroying the laminar dependence of the data. This realizes the null hypothesis that relative power/CFC/MUA values do not depend on laminar depth. A large-sample approximation of this permutation distribution is then used to compute the p-values we have reported (22). We obtained the same p-values when we manually created the permutation distributions using 10,000 permutations (but with this number of permutations could only resolve p values as low as  $1E-4$ ). We report p-values based on this large-sample approximation (22). All correlations were performed on un-smoothed data.

To assess the hypothesis that a given area's gamma/alpha-beta/CFC laminar profile was significantly correlated to the average of the remaining areas (Supplementary Figure 2 and 9), we used bootstrap resampling. This was necessitated by the fact that we wanted to correlate each area's average profile to an unbiased representation of the average of the remaining areas. To calculate an average of the remaining areas that was not biased by differences in sample size between areas, we resampled 10 laminar profiles from each area with replacement, and then took the average. This procedure was repeated 1,000 times to calculate a bootstrap distribution of Spearman rank correlation values between the average of each area and the (unbiased) average of the remaining areas. The reason we resampled with replacement was to approximate as closely as possible the classic bootstrap procedure (23). The reason we resampled 10 laminar profiles for each area was because the average number of laminar profiles per area was 10 in our study. Note that some areas were sampled less than 10 times, and

so the resampling for these areas will re-use the same laminar profile many times (in order to equally weigh the areas with less sampling in the average). Changing the number of area laminar profile resamples per bootstrap to 5 or 2 did not qualitatively change the results. We then used the formula from Efron and Tibshirani (23, equation 2.3) to calculate the standard error of the mean of this bootstrap distribution, and computed the 95% confidence intervals by multiplying this estimate of the standard error by 1.96. P-values for the bootstrap average correlation coefficient were determined by shifting the bootstrap distribution to zero by subtracting its mean. The p-value then corresponds to the rank of the average correlation coefficient relative to this zero-centered distribution divided by the number of bootstrap re-samples (24, equation 3.10).

## **Supplemental Results**

### **Analysis of isolated units and spike rates confirm delay-period activity in superficial layers**

Fig. S5A shows the number of times a laminar depth was sampled (in black), the number of units recorded at each depth (in green), and the number of units that were modulated (in blue), summed across sessions. Modulation was determined by testing whether each unit's firing rate during the delay was significantly different from its baseline firing rate by a two-tailed t-test at  $p < 0.01$ . Fig. S5B shows the proportion of units with delay activity (i.e., units modulated/units recorded). This was significantly different between superficial vs deep layers (Fig. S5B inset, 53% vs. 34%, Chi-squared proportion test,  $p = 3E-4$ ). The proportion of modulated units by layer (Fig. S5B) positively correlated with the gamma LFP laminar profile and negatively correlated with the alpha/beta LFP profile (Spearman rank correlation, gamma,  $\rho = 0.77$ ,  $p < 2E-4$ , alpha/beta,  $\rho = -0.67$ ,  $p = 0.002$ ). Spiking during the delay period carried significant information about the sample (measured using Percent Explained Variance, PEV, see Methods). The peak spike PEV value was observed in superficial layers at depth -400um, the same depth at which gamma power peaked. The more alpha/beta a layer contained, the less information it contained about the sample (Spearman rank correlation,  $\rho = -0.48$ ,  $p = 0.02$ , Fig. S6). The greater proportion of modulated units in superficial layers was not the result of a poor signal to noise ratio or lack of units in deep layers. In fact, baseline firing rates were higher in the deep layers (Fig. S5C), as was the unit yield (Fig. S5D). A similar delay-period profile was seen when we aligned depth based on the transition between cerebrospinal fluid and the gray matter (Fig. S7).

### **Sample onset and saccade processing activate spiking in middle and deep layers**

Anatomical and physiological studies of the cortical microcircuit have shown that feedforward inputs arrive in layer 4, and the output of cortical computation to motor structures is signaled via deep layers, especially layer 5 (15). Therefore, we investigated whether the sample processing and saccade execution stages of the task

would activate these other layers. For the granular cortical areas (VLPFC, DLPFC, 8A, sampled in monkeys S and P), the sensory MUA response at sample onset was strongest at depths near zero (depth -200  $\mu\text{m}$  for monkey S, 100  $\mu\text{m}$  for monkey P, Fig. S8 A/E). In addition, all areas/monkeys showed robust deep layer activation just preceding the saccade (Fig. S8 B/D/F/H). The ratio of MUA activity in deep layers to superficial layers was significantly larger in the 50 ms prior to the saccade compared to the delay period (sign test,  $p < 0.01$ , Fig. S8H/I). This indicates that the relative balance in cortex switches from superficial-layer-dominant during the delay to driven by both deep and superficial layers during the saccade period.

### **Recurring dynamics across all six cortical areas**

To test the hypothesis that certain aspects of the laminar dynamics are shared across areas, we calculated each area's average gamma and alpha/beta relative power profile and each area's cross-frequency coupling (CFC) profile between deep layers' alpha/beta phase to all other layers' gamma amplitude. Each area's average profile was taken across all available data from that area, and is shown as the bold lines in the upper subpanels of Fig. S2 and S9. We reasoned that if there are shared dynamics, then the average of each area should be correlated to the average of the remaining areas. To test this, we calculated Spearman rank correlation between each area's average power/CFC profile and the average of the remaining areas (excluding the current area, so as to perform correlation on independent data, and equally weighing each area when computing the other-area average, see Analysis, statistics). We assessed significance of the correlation using a bootstrap resampling procedure (see Analysis, statistics). In SI Figures 2 and 9, we plotted these bootstrap distributions and their 95% confidence intervals. In SI Figure 2, the gamma and alpha/beta relative power profiles of each area are significantly correlated to the unbiased other-area-average (Bootstrap test,  $p < 0.01$ ) in all cases except one (alpha/beta profile of area 8B). In SI Figure 9, the CFC profile of each area is significantly correlated to the unbiased other-area-average in all cases (Bootstrap test,  $p < 0.01$ ).

Finally, we determined the significance of observing the peak delay-period MUA modulation in superficial areas for all areas under the null hypothesis that the peak could occur in either superficial or deep layers with a sign test over the six areas ( $p = 0.03$ , SI Figure 3).

### **Spiking and bursting are modulated on error trials**

To add evidence for the functional relevance of our measures of MUA modulation and LFP bursts, we analyzed the error trials. Compared to error trials, correct trials had elevated MUA in deep layers (sign test,  $p < 0.05$ , Bonferroni corrected, Fig. S11A). Correct trials were associated with less LFP gamma bursting during the delay (sign test,  $p < 0.05$ ) but more alpha/beta bursting (sign test,  $p < 0.05$ ) during the baseline (Fig. S11B). These results indicate that the strength of delay period MUA modulation and the strength of alpha/beta and gamma bursting are relevant for correct WM performance.

These results suggest that both superficial and deep layers are important for correct WM performance, but in distinct ways. As we show in Fig. 4B, gamma bursting during the delay in superficial layers is informative about the sample (in correct trials). Deep layer alpha/beta rhythms modulate this gamma (Fig. 5B). This deep to superficial modulation has an inhibitory effect on superficial gamma (Figure 5E). Thus, errors could come about by a combination of mechanisms: less (inhibition-related) alpha/beta bursting during the baseline would make the default state of the cortex overly excited with internal information, and less receptive to the sample (on error trials). This is born out in the delay, where we observe an excessive amount of gamma bursting in error trials, and a relative lack of deep layer MUA. Thus, we interpret error trials as a failure of control-related deep layer activity. This results in aberrant modulation of WM information-related gamma.

## **Supplemental Discussion**

### **Sustained activity and Working Memory**

In all three tasks that we studied, we observed changes in MUA activity during the delay vs. baseline. Two of the tasks (involving spatial WM) also involved maintaining a motor plan, while the object WM task did not. In all cases, we observed modulation in average MUA activity in superficial layers. This is evidence that the MUA modulation is not a trivial consequence of oculomotor signals (which are not informative in the delay period of the object based task). This suggests that neuronal activity is regulated during WM delays, although not necessarily in a sustained pattern of firing. Indeed, recent models suggest that WM information is encoded in a pattern of synaptic strength, and is periodically refreshed during the occurrence of gamma oscillations (20, 21). Thus, elevated spiking is not necessary throughout the trial. Our finding of a strong correlation between layers with gamma and layers with delay activity supports this model, and suggests that this mechanism occurs predominately in superficial layers.

## **Supplemental Figures Captions**

**Supplemental Figure 1.** **A.** The grand average current source density analyses calculated with respect to sample onset (during working memory task performance) or **B** after flash onset (during pre-task mapping) and after alignment to the first significant sink on each electrode. The white contour lines reflects significance at  $p < 2.4E-5$  (Bonferroni correction). **C.** Power asymmetry in the gamma and alpha/beta bands calculated based on data epochs from 200ms pre-sample onset to 500ms post-sample onset (during task performance). **D.** Power asymmetry in the gamma and alpha/beta bands calculated based on data epochs from 200ms pre-flash onset to 500ms post-flash onset (during pre-task mapping). Both gamma and alpha/beta profiles in **C** and **D** have a Spearman correlation of 1 ( $P < 5E-7$ ).

**Supplemental Figure 2.** (Upper subpanels) Relative power profiles in the gamma (red, thick lines) and alpha/beta (blue, thick lines) bands across areas, in order (left to right

and top to bottom) of areas with more to less sampling (N, which is the number of laminar probes per area). The less saturated and thinner red/blue lines are the bootstrapped re-sampled and bias-free average of the remaining areas (other-area averages were corrected for the differences in N between areas). The solid and thin lines for each area were correlated, and the average (over bootstrap resamples) resulting of Spearman rank correlation (Rho) and their p-values are plotted above each upper subplot. (Lower subpanels) The distribution of bootstrap resampled Spearman's Rho between each area's average power profile and the unbiased-other-area average are plotted separately for alpha/beta (blue) and gamma (red). The 95% confidence interval for these distributions are shown in the thick red/blue lines above the distribution. For the areas with sparse N (bottom row), the y-axis has been adjusted to show laminar depths which were consistently recorded in that area.

**Supplemental Figure 3.** The average delay-period MUA modulation per cortical depth across all areas we sampled, in order (left to right and top to bottom) of areas with more to less sampling. For the areas with sparse sampling (bottom row), the y-axis has been adjusted to show laminar depths which were consistently recorded in that area.

**Supplemental Figure 4.** A representative session's peri-stimulus time histogram (PSTH) depicting average firing rate  $\pm$  1SEM in dotted lines across the trial for the masked delayed saccade task. The task progression (fixation, sample onset, mask onset, delay) is shown in the upper PSTH. Four units are shown (2 in superficial layers, 2 in deep layers) along with their depth on the laminar probe, indicated by the arrows. Above each unit's PSTH is the unit's unique identifying code and the number of trials for which the respective unit was observed. To the right of each PSTH, a random sample of 1,000 individual spike waveforms are shown for each respective unit (the total number of spikes for which the unit was observed is depicted above each waveform).

**Supplemental Figure 5. A.** Number of times a depth was sampled (in red), number of units detected (in green) and the absolute number of units significantly modulated (in blue) during the delay (t-test,  $p < .01$ ) across laminar depths. **B.** The proportion of units that were significantly modulated during the delay across depths. (inset) The proportion modulated in superficial vs. deep layers. Error bars  $\pm$  1 SEM. **C.** The mean baseline firing rates across laminar depths. **D.** The proportion of electrodes with detectable units.

**Supplemental Figure 6.** Power asymmetry in the gamma (red) and alpha/beta (blue) bands over laminar depth (same as in Figure 2B). In black, the information (calculated with PEV) in delay-period spiking about the sample over laminar depth is plotted.

**Supplemental Figure 7.** The proportion of units that were significantly modulated during the delay for each laminar depth, aligned to the transition zone between cerebrospinal fluid (CSF) and the gray matter (depth 0). This transition zone was estimated as the contact where a sudden drop in power of 70% or more across all LFP frequencies occurred. This was only possible in recording sessions where at least one channel remained outside the cortex in CSF (N=24 sessions). For this subset of

sessions, the proportion of units with delay activity showed a similar profile as the full dataset, peaking in more superficial layers, and decreasing with depth.

**Supplemental Figure 8.** *A/C/E (left)*. Average MUA modulation with respect to fixation baseline, time-locked to sample onset and plotted over time and laminar depth, for each respective task. *A/C/E (right)*. Same as *A/C/E (left)*, but average over the time window indicated above the graphs, from 50-100ms post sample onset (in black) and for the first 1 second of the delay period (in blue). *B/D/F (left)*. Average MUA modulation with respect to fixation baseline, time-locked to saccade execution and plotted over time and laminar depth, for each respective task. *B/D/F (right)*. Same as *B/D/F (left)*, but average over the time window indicated above the graphs as the saccade window from 0-50ms pre-saccade (in red) and the end of the delay period (in blue).

**Supplemental Figure 9.** (Upper subpanels) The average delay-period Cross-Frequency Coupling of each area (thick blue lines). The Cross-Frequency Coupling was quantified using the modulation index (see Methods). The modulation index profile was calculated by averaging across deep layers' (depths greater than +100 um alpha/beta phase) alpha/beta phase, and plotting the resulting CFC modulation of the gamma amplitude of all other layers. Areas are plotted in order (left to right and top to bottom) with more to less sampling (N, which is the number of laminar probes per area). The thinner gray lines are the bootstrapped re-sampled and bias-free average of the remaining areas (other-area averages were corrected for the differences in N between areas). The solid and thin lines for each area were correlated, and the average (over bootstrap resamples) resulting of Spearman rank correlation (Rho) and its p-value are plotted above each upper subplot. (Lower subpanels) The distribution of bootstrap resampled Spearman's Rho between each area's average CFC profile and the unbiased-other-area average is plotted. The 95% confidence interval for these distributions are shown in the thick black lines above the distribution. For the areas with sparse N (bottom row), the y-axis has been adjusted to show laminar depths which were consistently recorded in that area.

**Supplemental Figure 10.** **A.** Cross Frequency Coupling (CFC) between the phase of alpha/beta oscillations and the amplitude of gamma oscillations during the baseline period. Plotted across both axes is the CFC between specific cortical depths. **B.** The mean CFC across all four possible conditions during the baseline period: superficial phase to superficial amplitude (left), deep phase to deep amplitude (middle-left), superficial phase to deep amplitude (middle-right), and deep phase to superficial amplitude (right).

**Supplemental Figure 11.** **A.** The difference between the delay-period MUA modulation of correct trials minus error trials per laminar depth, averaged across sessions with at least 20 error trials (N=56). Error bars +/- 1 SEM for illustration. The asterisks denotes the depths at which the differences were significant at  $P < 0.05$  (sign test across sessions, red, Bonferonni corrected for multiple comparisons, black uncorrected). **B.** The average bursting rate across layers and sessions in the alpha/beta (left) and gamma (right) bands, during the baseline fixation period (upper graphs) and during the

delay period (lower graphs). Error bars +/- 1 SEM for illustration. Asterisks denotes significant differences at  $P < 0.05$  (sign test over sessions).

**Supplemental Figure 12.** **A.** Laminar profile of gamma and alpha/beta LFP over recording depth in area SMA. In this case, layers are inverted with respect to channel depth: deep cortical layers (L5/6) are in more superficial channel depths, and superficial cortical layers (L2/3) are in deeper channel depths. **B.** Laminar profile of gamma and alpha/beta LFP over layers in ACC: the power profile re-normalizes, with superficial gamma in superficial channel depths, and deep alpha/beta in deep channel depths. **C.** An MRI slice of frontal cortex, aligned to the recording grid (white lines). The red lines and arrow correspond to a continuation of the white lines, and therefore indicate approximate recording trajectories. Recording trajectories were chosen to be as perpendicular as possible to cortex. The left most trajectory could also be used to access the deeper cortical areas (SMA and ACC). Area labels are approximate and based on (1) - coronal slice +28mm rostral to Ear Bar Zero. The MRI is in the same plane as the recording grid, therefore the sulcus to the right of the trajectories corresponds to the genu of the arcuate sulcus. **D.** The recording sites used in this study relative to anatomical landmarks. The dotted black line corresponds to the sample trajectories shown in **C.** **E.** Laminar profile of gamma and alpha/beta LFP over recording depth in area PMd for a representative session.

### **Supplemental Tables**

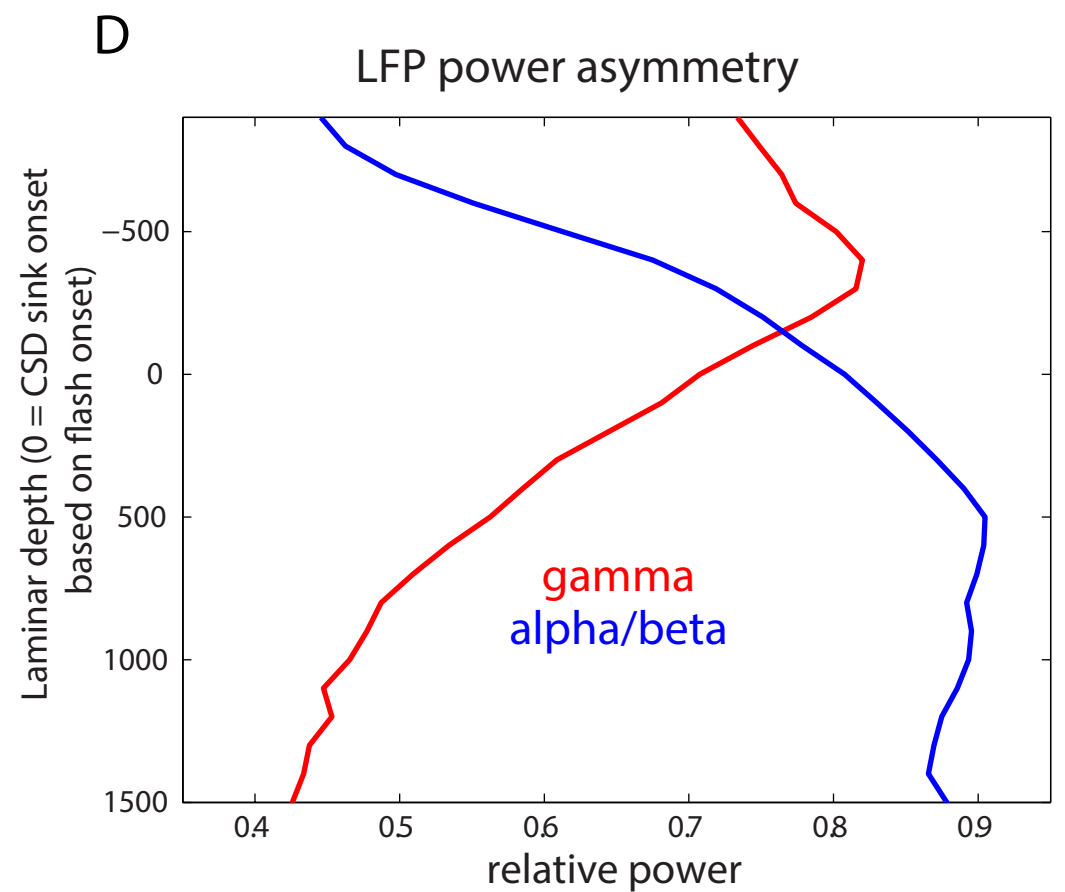
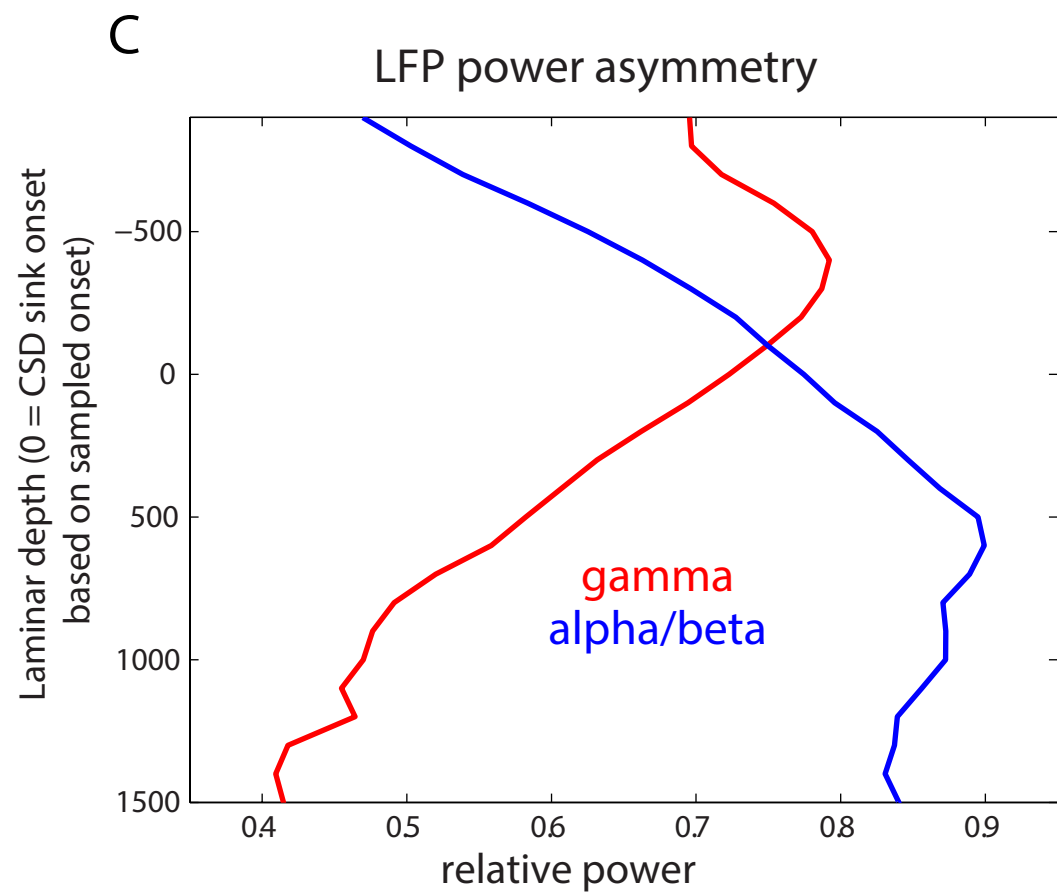
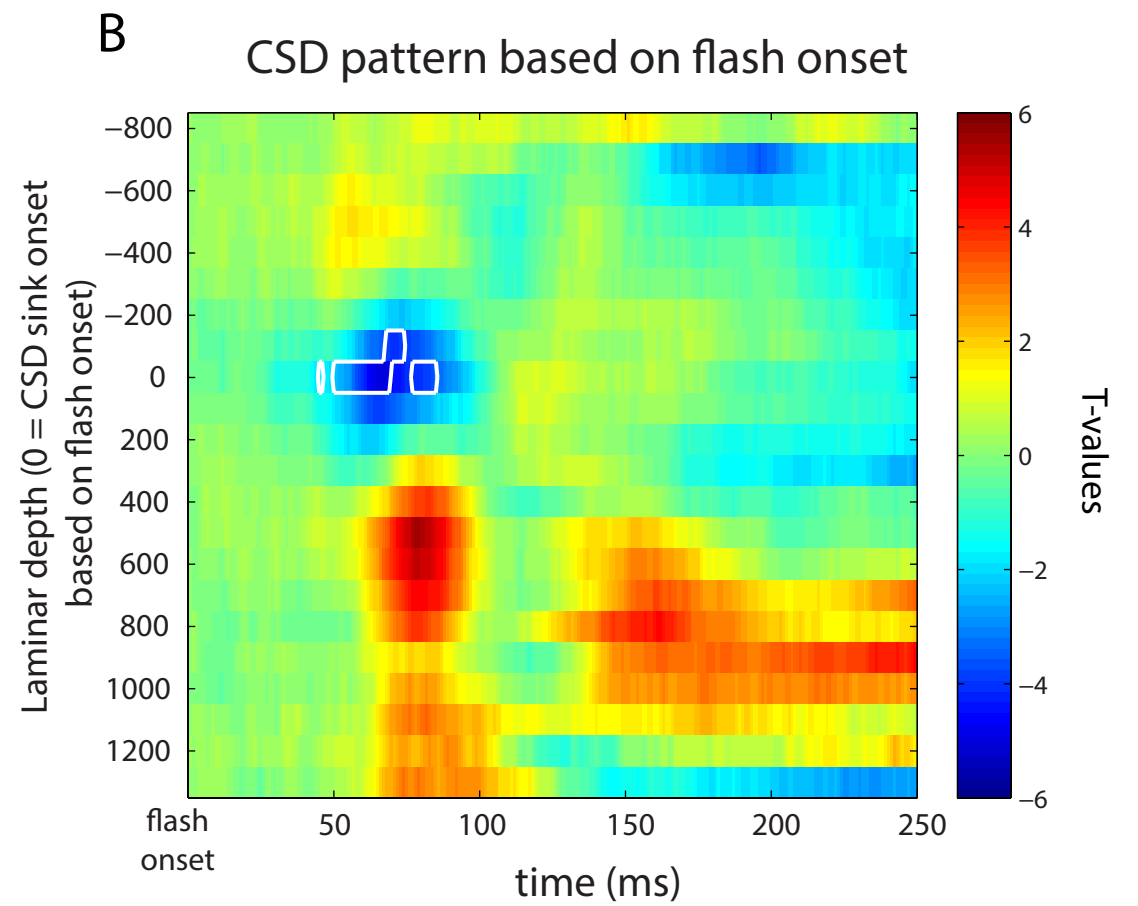
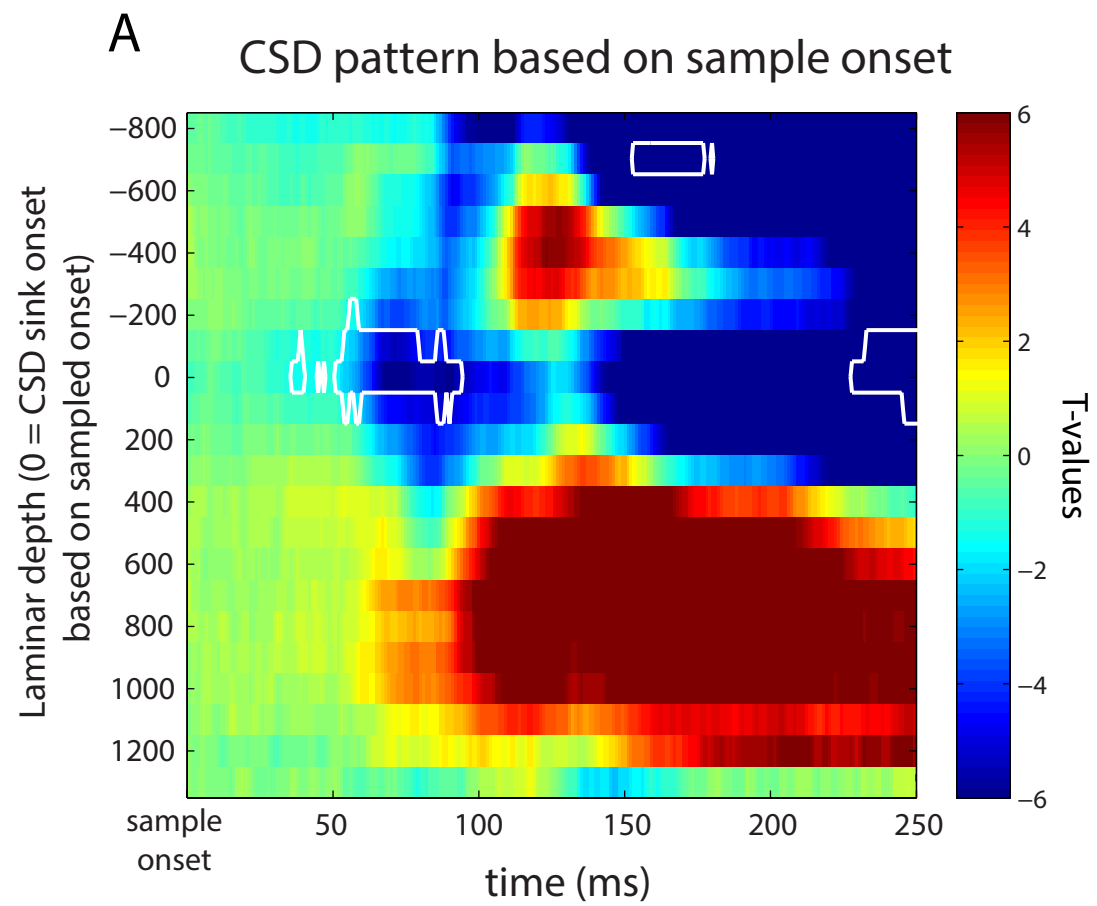
**Supplemental Table 1.** A table listing information about each of the 60 recordings we performed. From left to right, we list the number of channels on the U/V probe, the inter-electrode distance, the total coverage of the probe, the cortical area sampled, the monkey recorded, and the task performed.



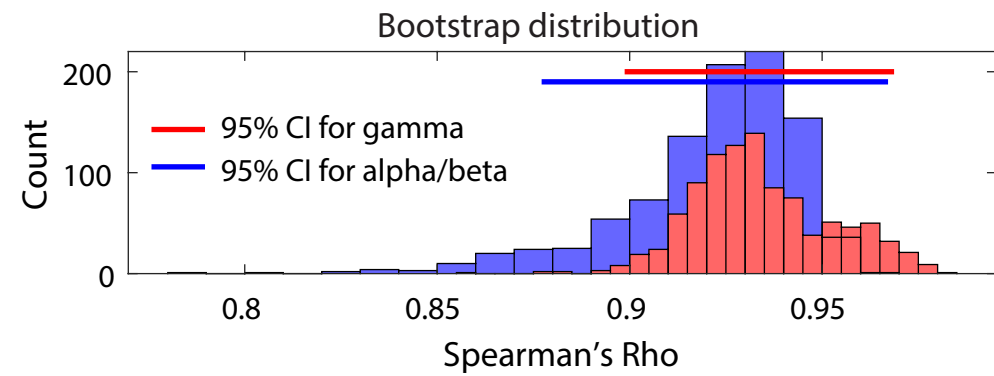
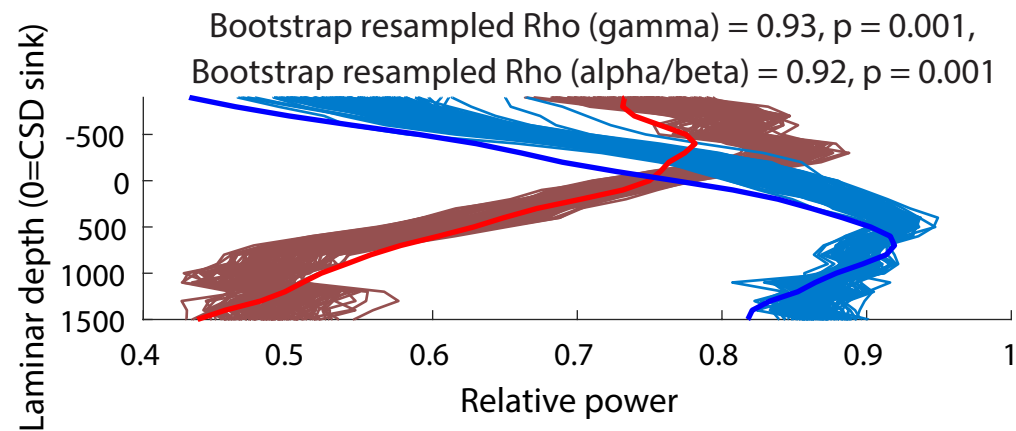
**Supplemental Table 1**

| Num channels on probe | Spacing (um) | Coverage (um) | Area  | Monkey | Task                   |
|-----------------------|--------------|---------------|-------|--------|------------------------|
| 16                    | 200          | 3000          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 150          | 2250          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 150          | 2250          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 200          | 3000          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 200          | 3000          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 150          | 2250          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 200          | 3000          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 150          | 2250          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 200          | 3000          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 150          | 2250          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 200          | 3000          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 200          | 3000          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 150          | 2250          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 200          | 3000          | ACC   | C      | Masked Delayed Saccade |
| 16                    | 150          | 2250          | ACC   | C      | Masked Delayed Saccade |
| 16                    | 200          | 3000          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 150          | 2250          | SMA   | C      | Masked Delayed Saccade |
| 16                    | 200          | 3000          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 200          | 3000          | 8B    | C      | Masked Delayed Saccade |
| 24                    | 150          | 3450          | 8B    | C      | Masked Delayed Saccade |
| 32                    | 100          | 3100          | PMd   | C      | Masked Delayed Saccade |
| 32                    | 100          | 3100          | PMd   | C      | Masked Delayed Saccade |
| 32                    | 100          | 3100          | SMA   | C      | Masked Delayed Saccade |
| 32                    | 100          | 3100          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 200          | 3000          | 8B    | C      | Masked Delayed Saccade |
| 16                    | 150          | 2250          | PMd   | C      | Masked Delayed Saccade |
| 16                    | 150          | 2250          | PMd   | C      | Masked Delayed Saccade |
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| 16                    | 200          | 3000          | VLPFC | S      | Search                 |
| 16                    | 200          | 3000          | VLPFC | S      | Search                 |
| 16                    | 200          | 3000          | 8A    | S      | Search                 |
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| 16                    | 200          | 3000          | 8A    | S      | Search                 |
| 16                    | 200          | 3000          | 8A    | S      | Search                 |
| 16                    | 200          | 3000          | VLPFC | S      | Search                 |
| 16                    | 200          | 3000          | 8A    | S      | Search                 |
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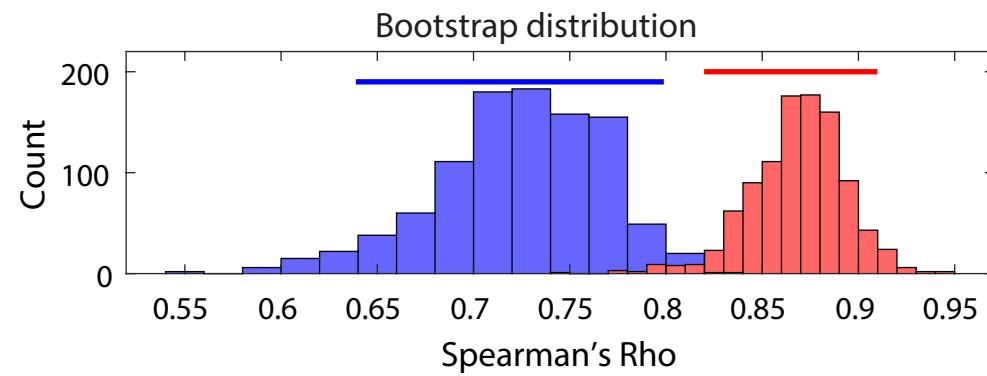
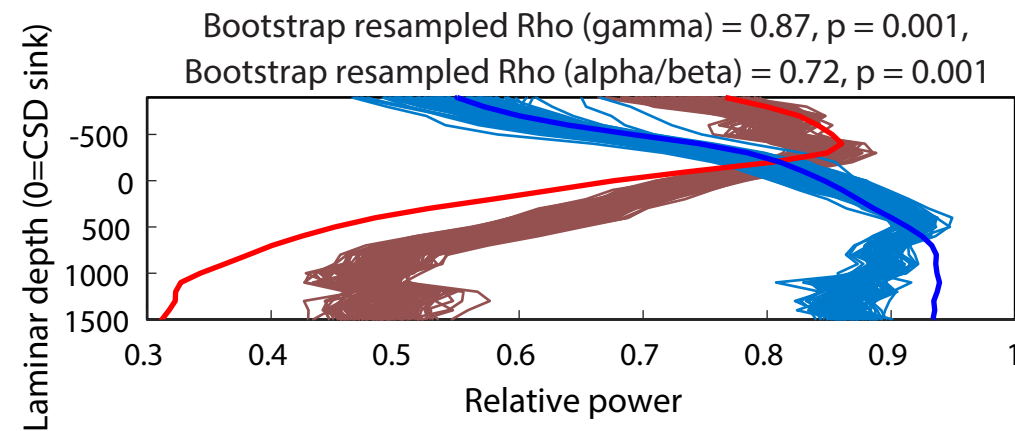
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| 16 | 200 | 3000 | 8A    | S | Search          |
| 16 | 200 | 3000 | 8A    | S | Search          |
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| 16 | 200 | 3000 | VLPFC | S | Search          |
| 16 | 200 | 3000 | VLPFC | S | Search          |
| 16 | 200 | 3000 | VLPFC | S | Search          |
| 16 | 200 | 3000 | VLPFC | S | Search          |
| 16 | 200 | 3000 | VLPFC | S | Search          |
| 32 | 100 | 3100 | VLPFC | P | Delayed Saccade |
| 32 | 100 | 3100 | VLPFC | P | Delayed Saccade |
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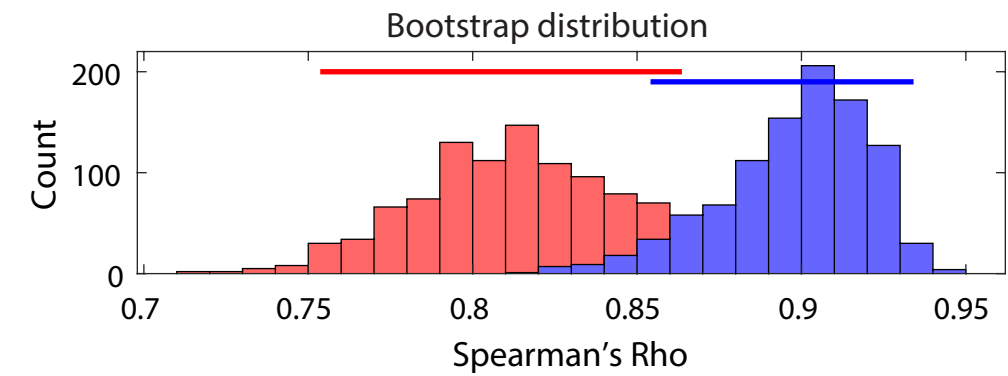
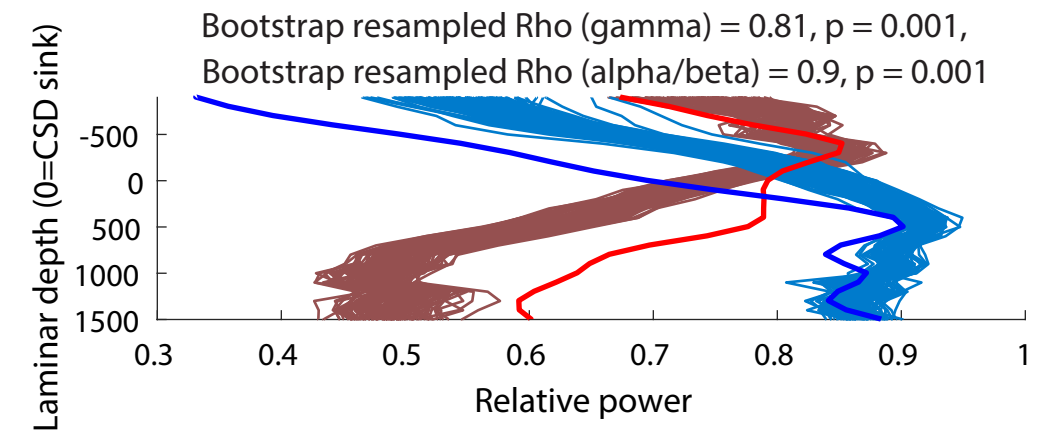
### VLPFC (N=23)



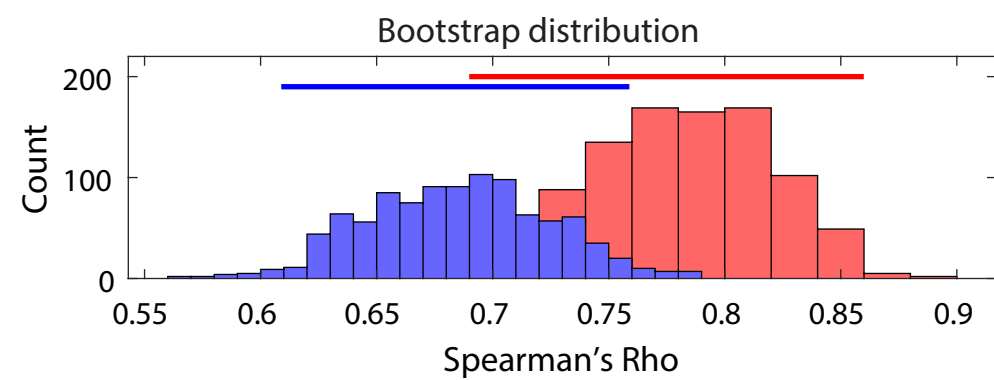
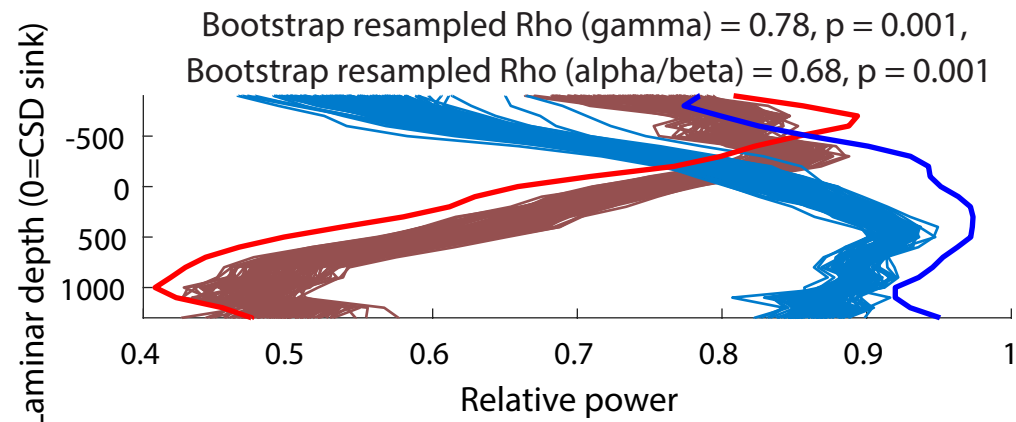
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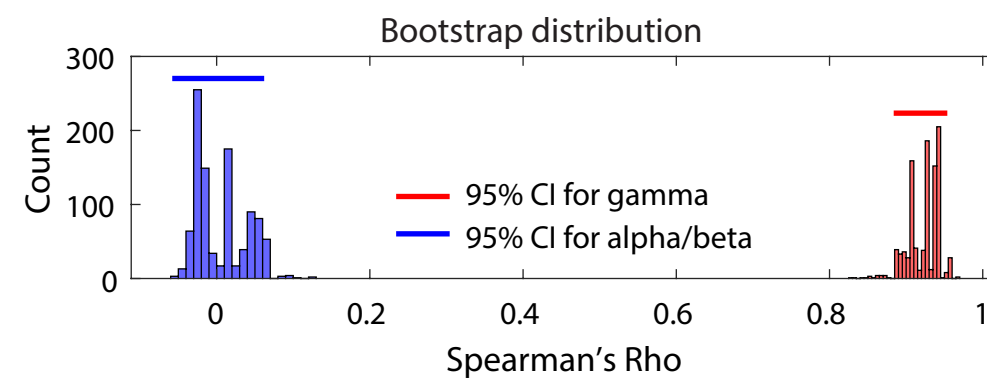
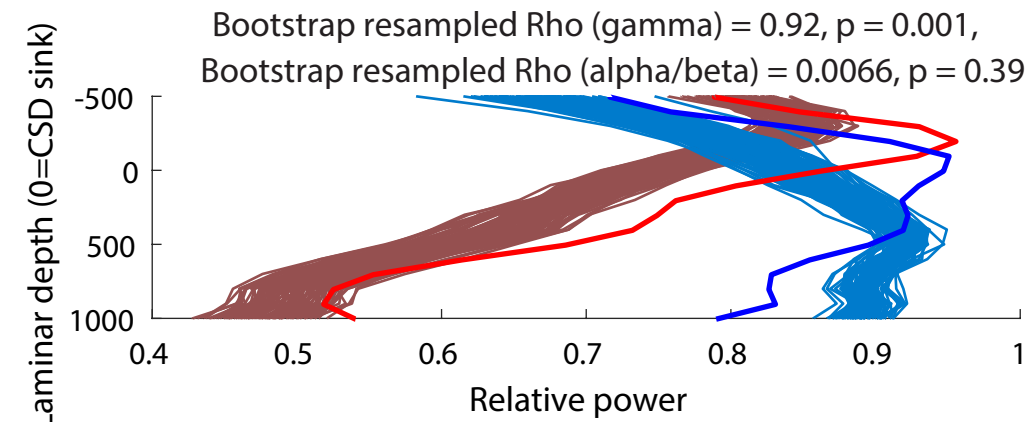
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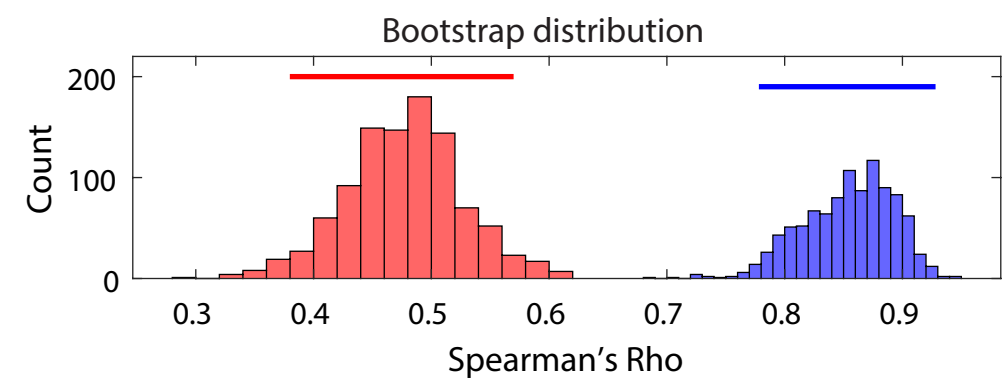
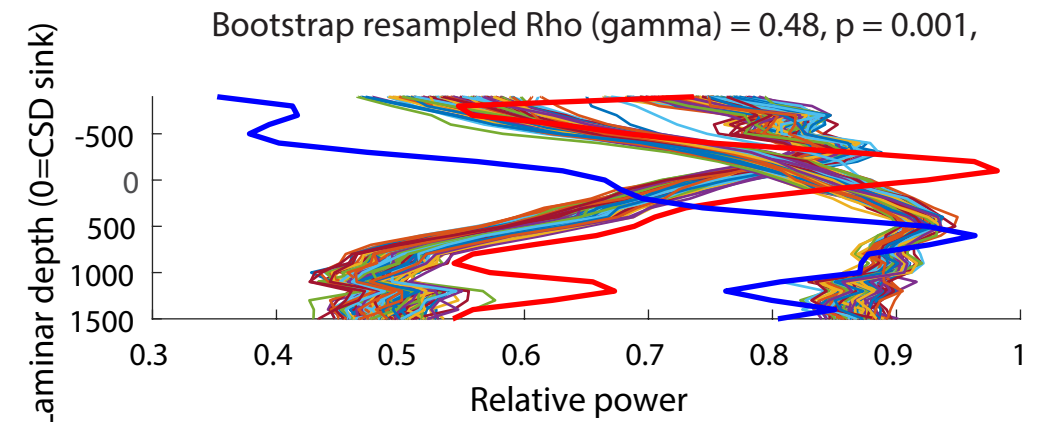
### ACC/SMA (N=4)



### 8B (N=3)



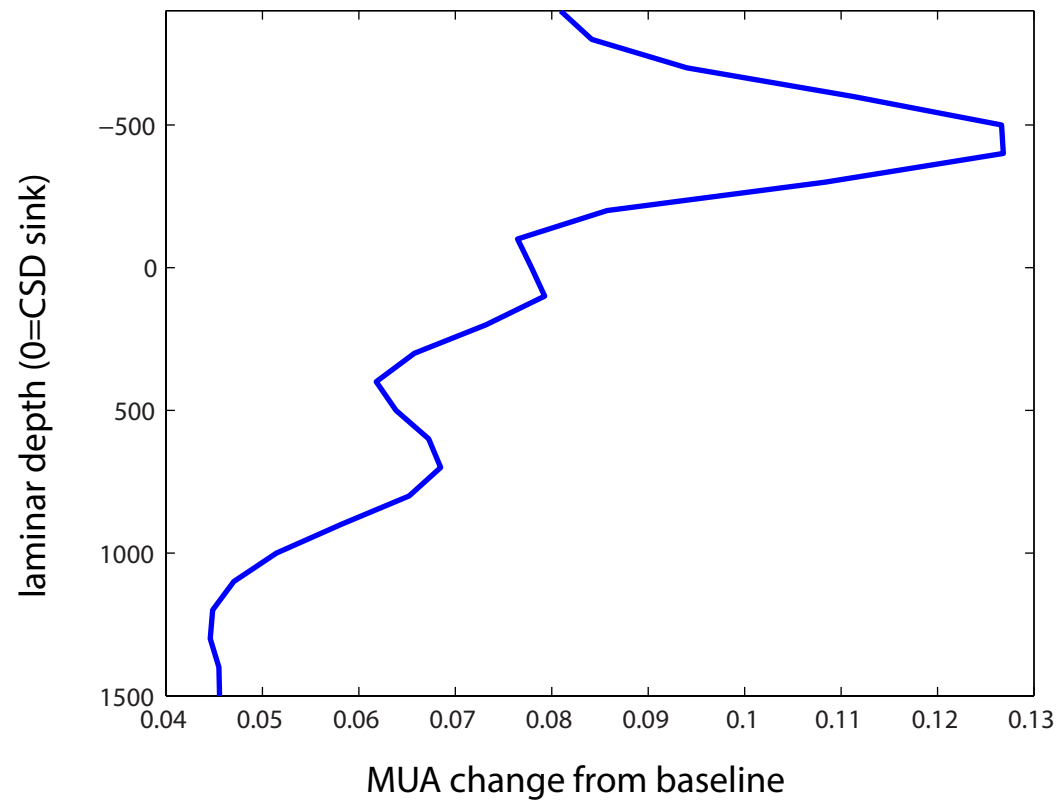
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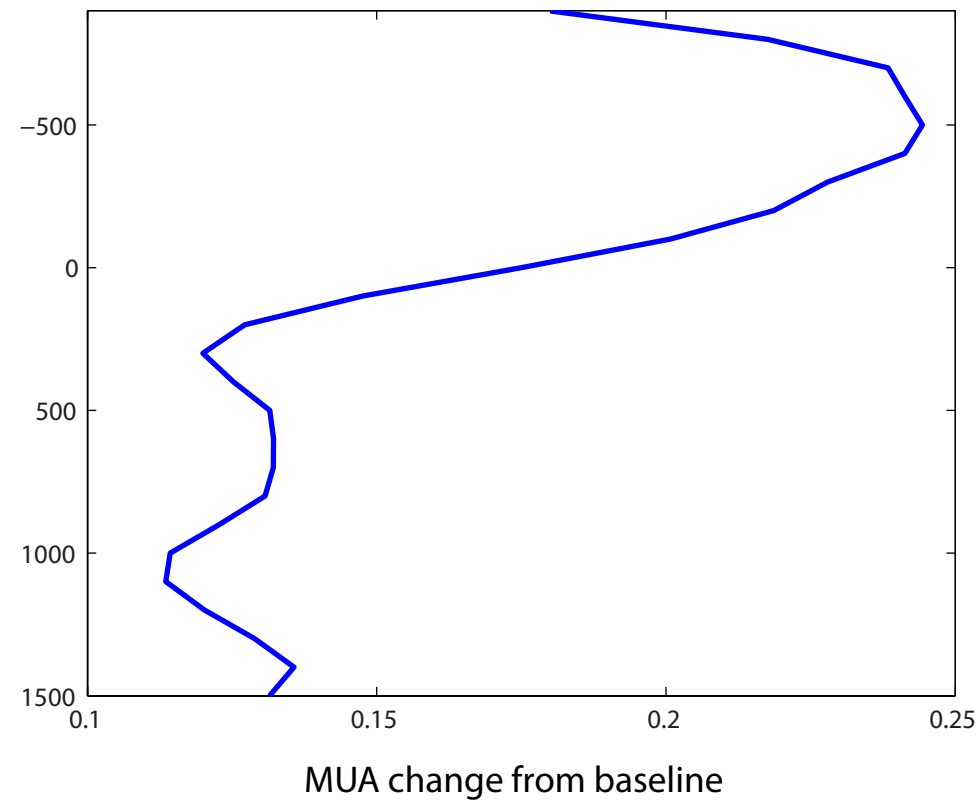
#### Legend (upper subplots)

- Gamma power (average of each area)
- Alpha/beta power (average of each area)
- Gamma power (resampled average of all other areas)
- Alpha/beta power (resampled average of all other areas)

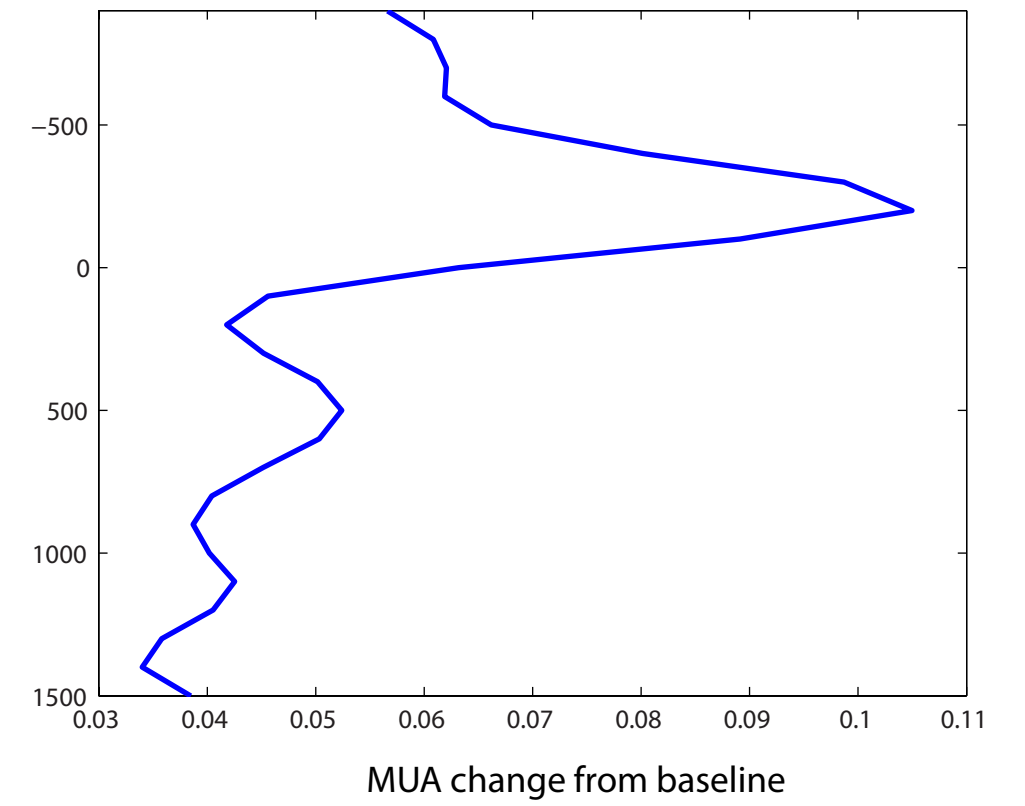
VLPFC (N=23)



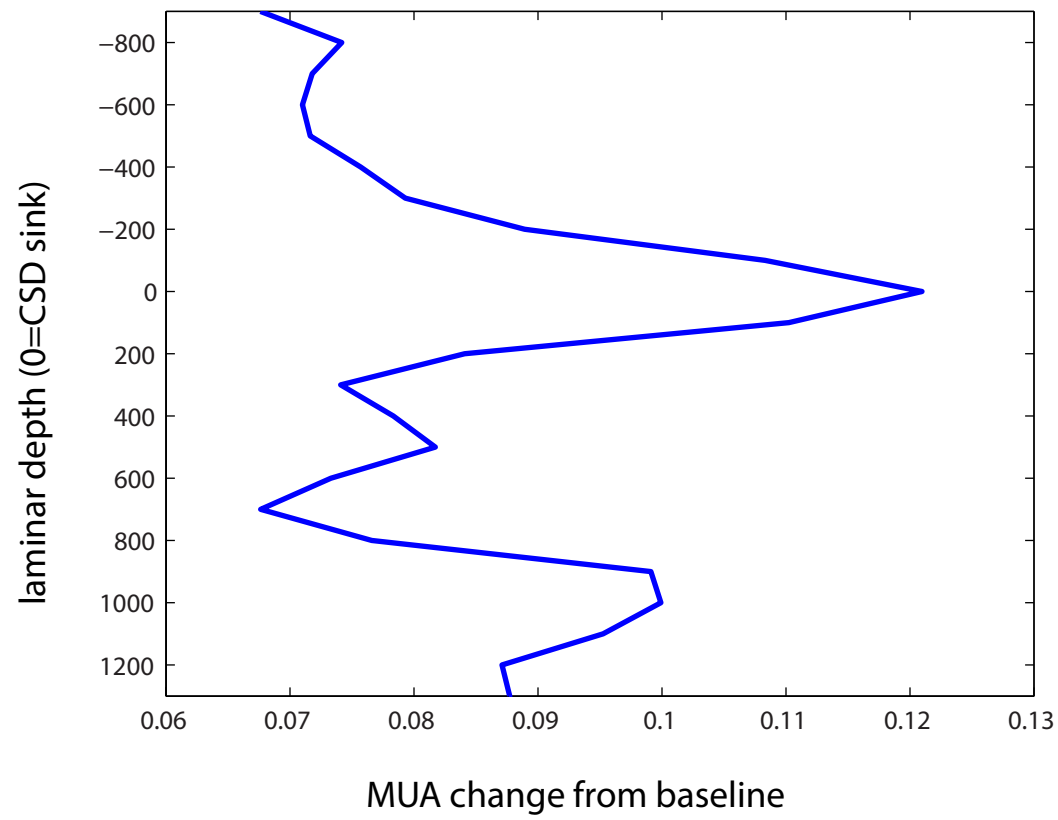
PMd (N=20)



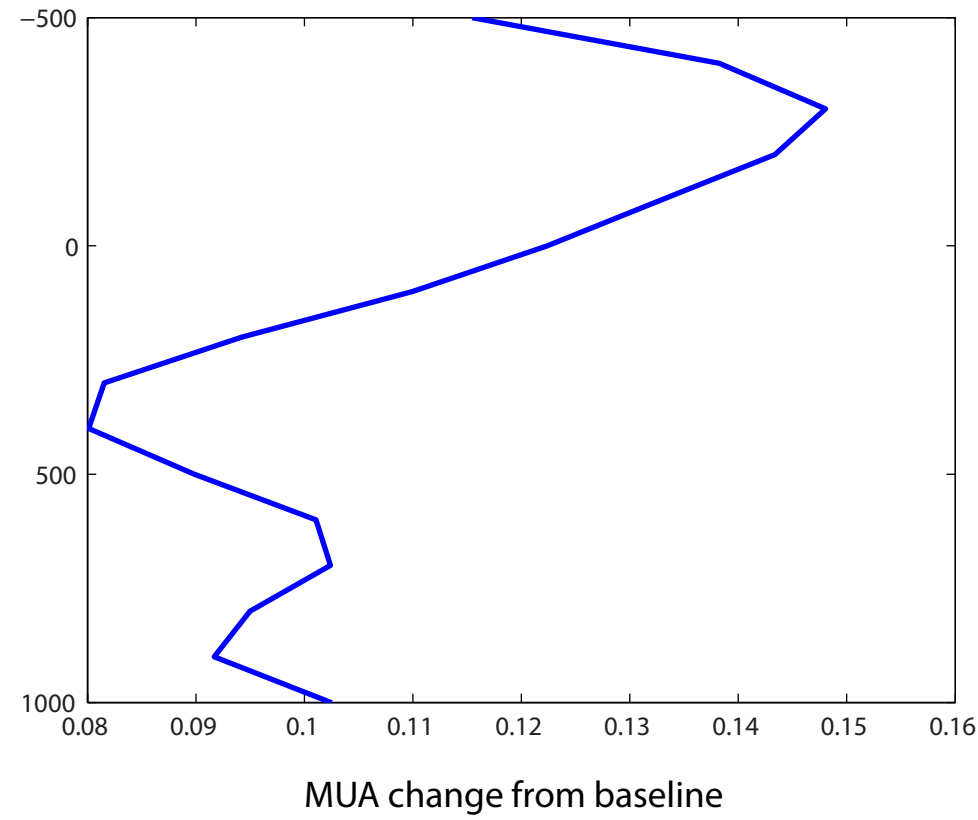
8A (N=8)



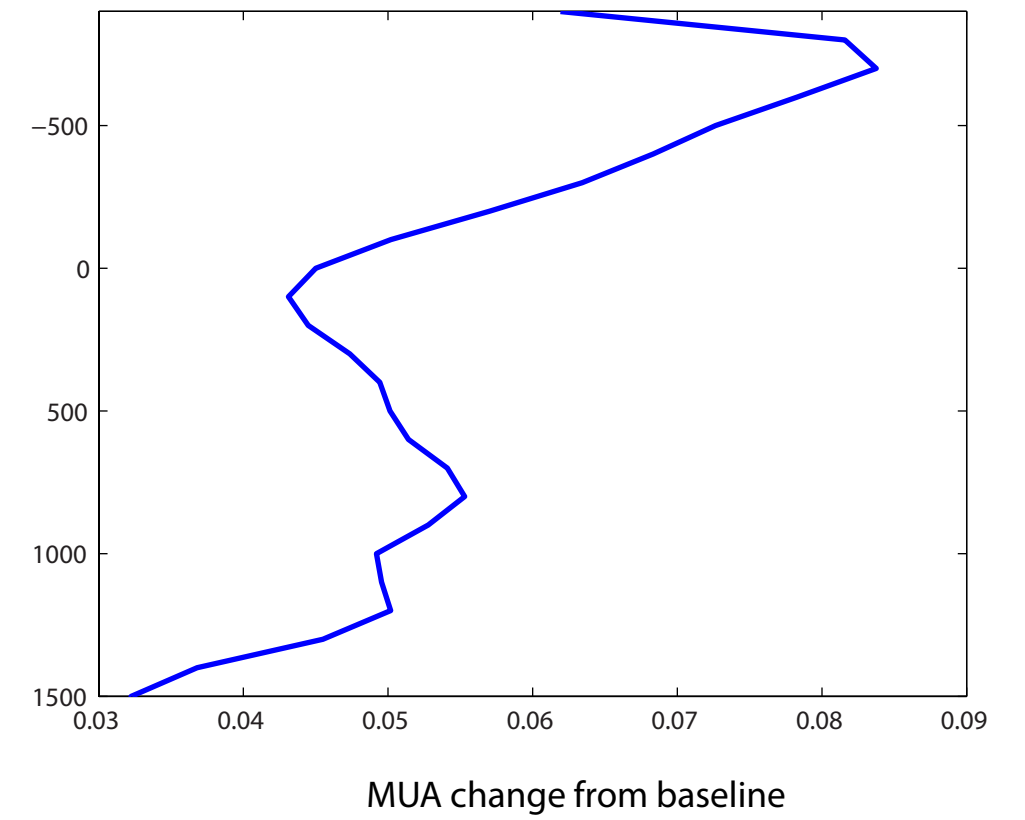
ACC/SMA (N=4)



8B (N=3)

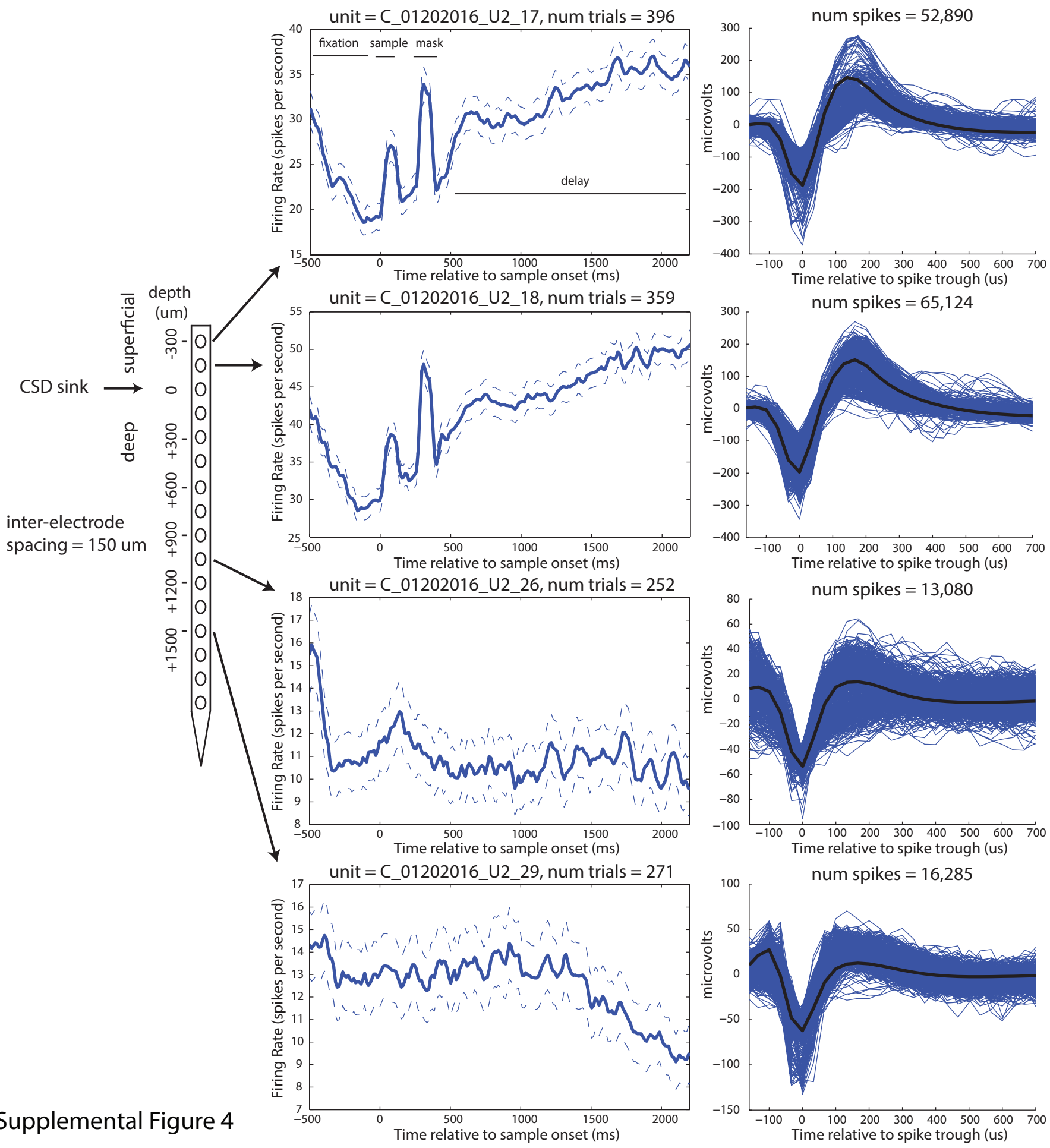


DLPFC (N=2)

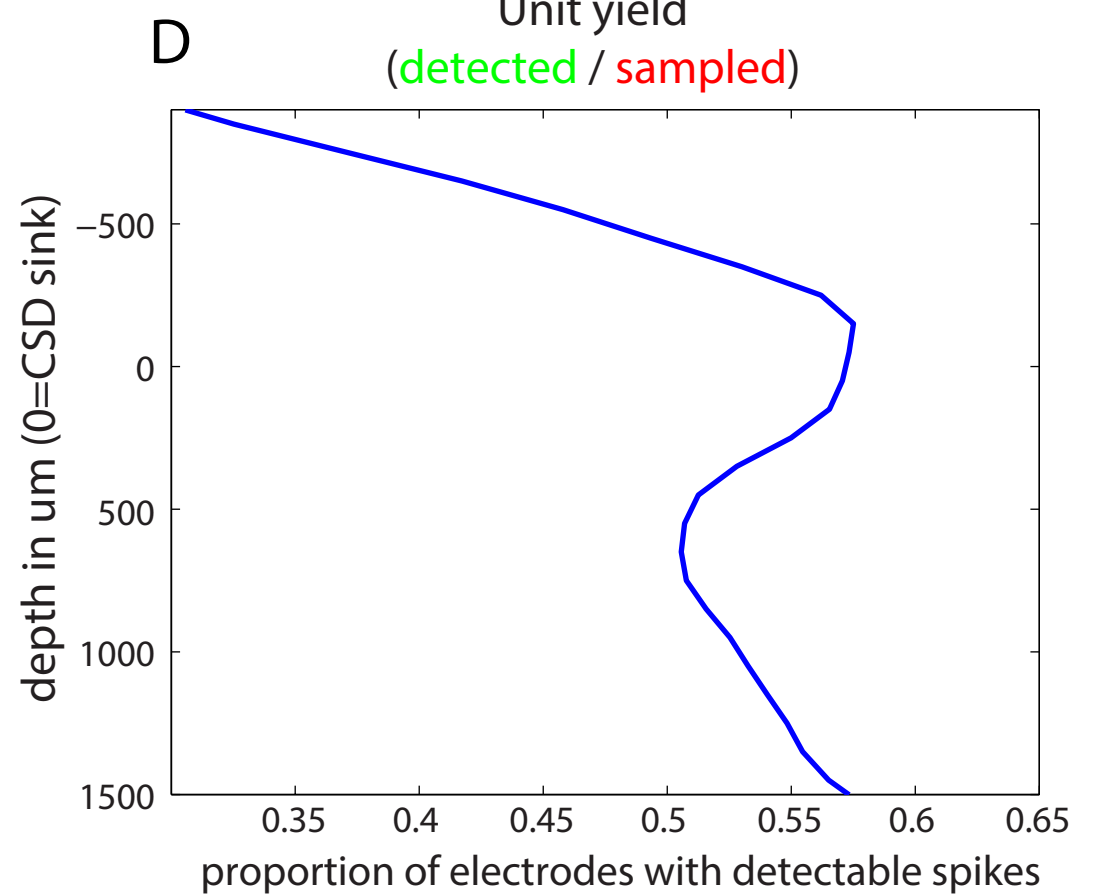
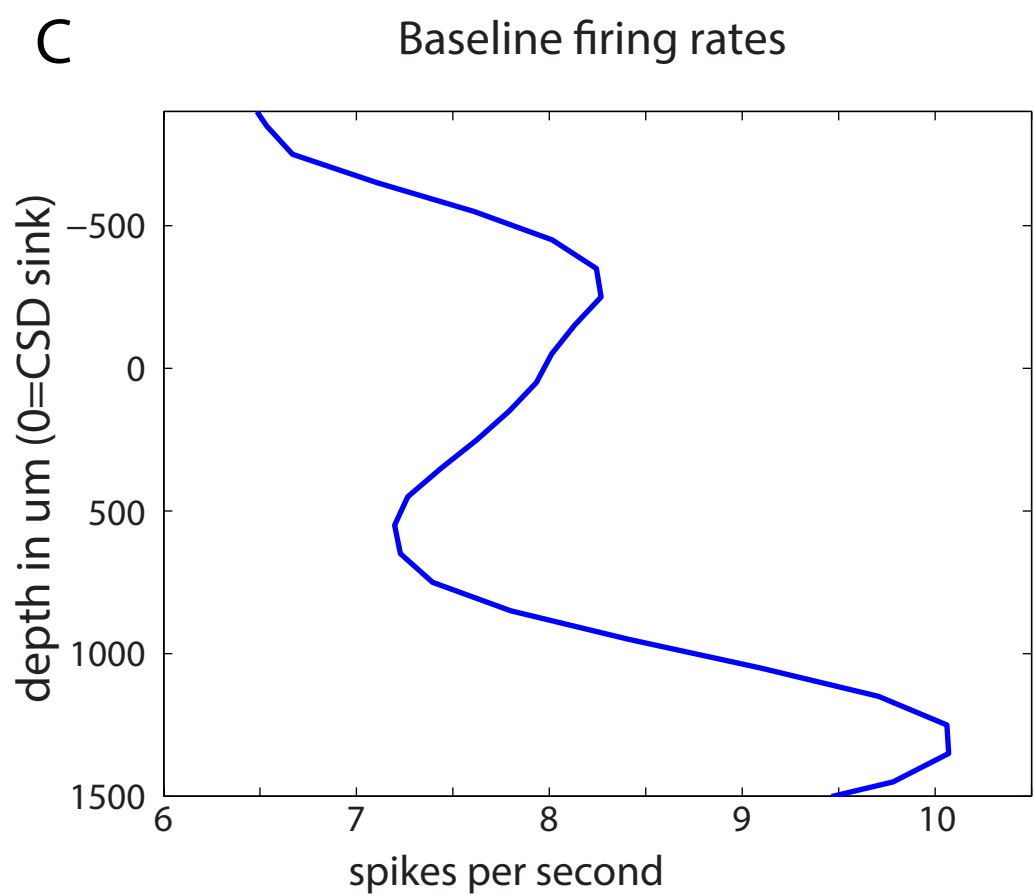
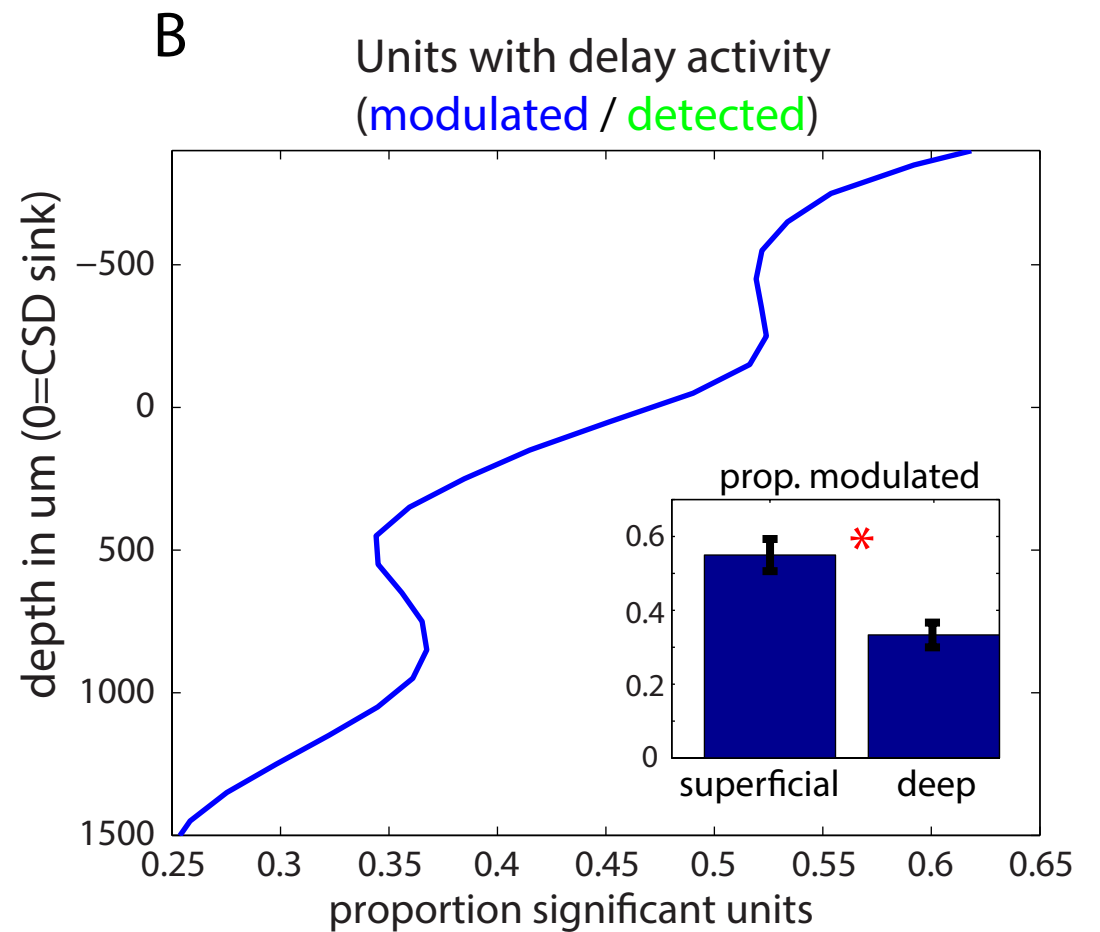
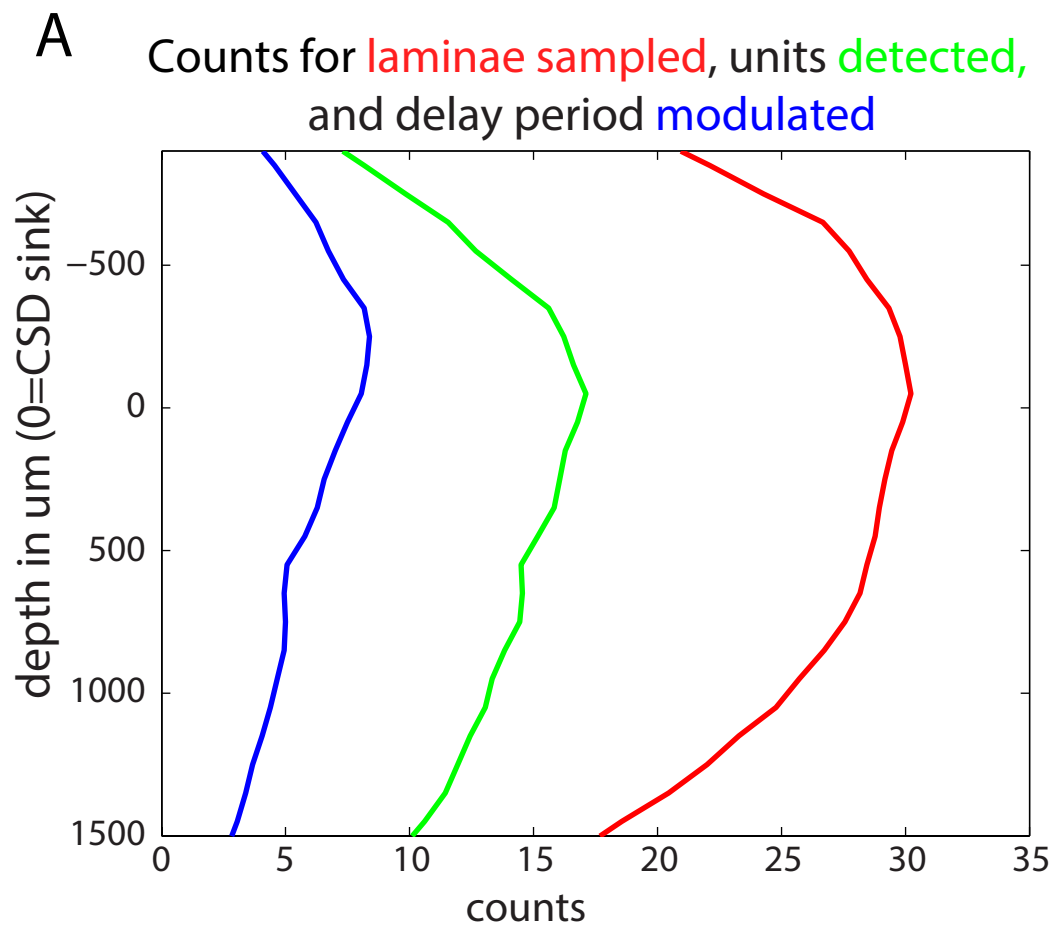


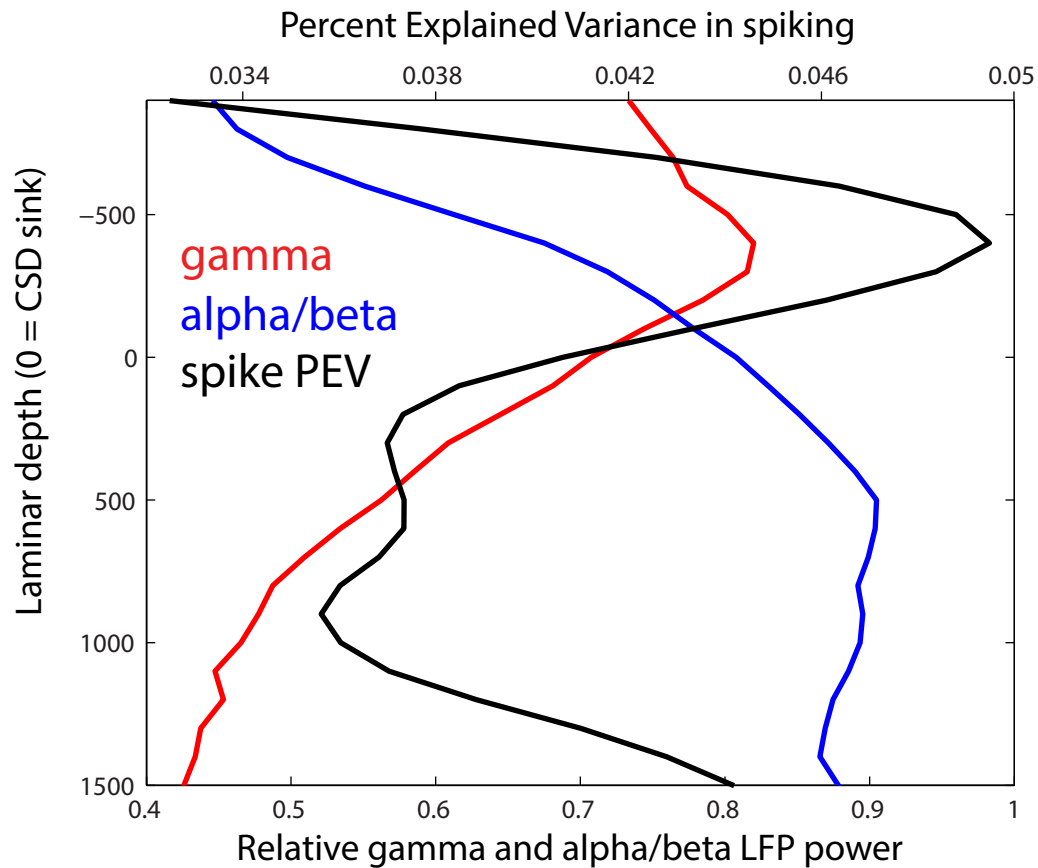
**Legend**

— Delay MUA (average of each area)



Supplemental Figure 4

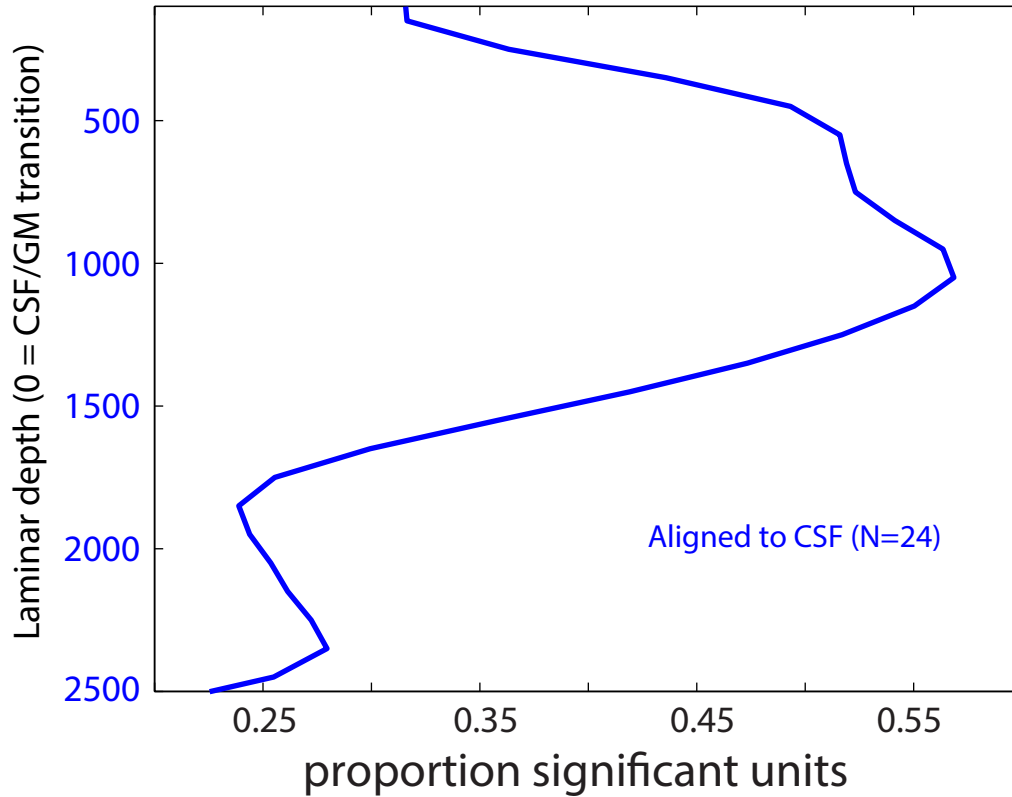




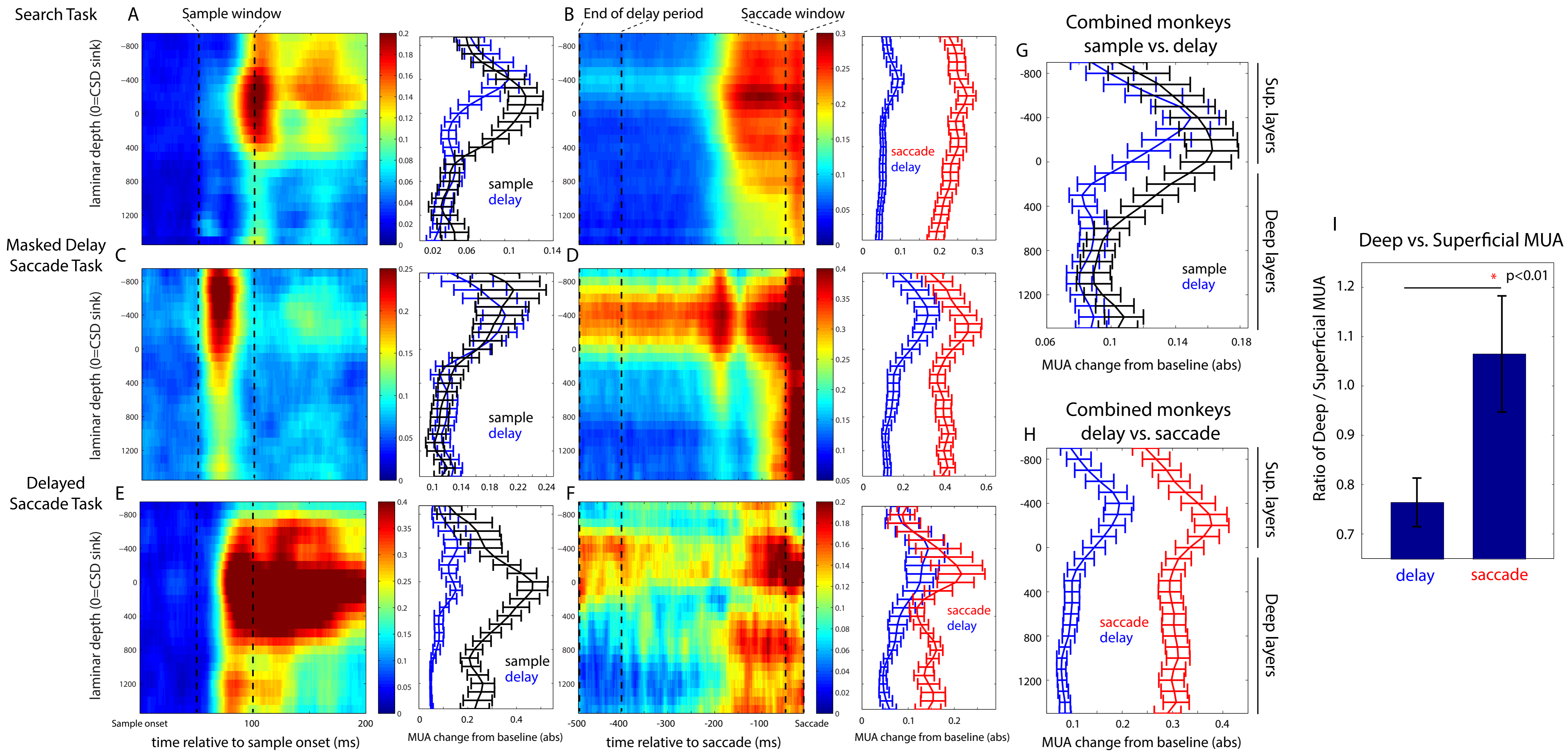
Supplemental Figure 6



# Units with delay activity

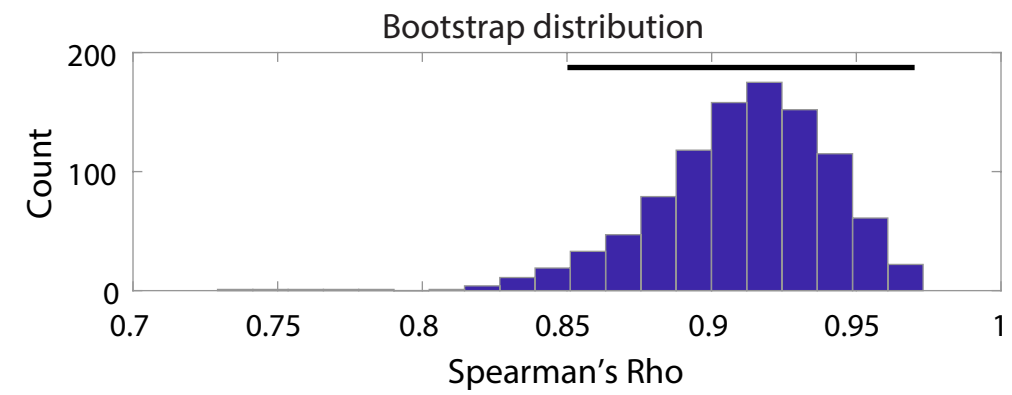
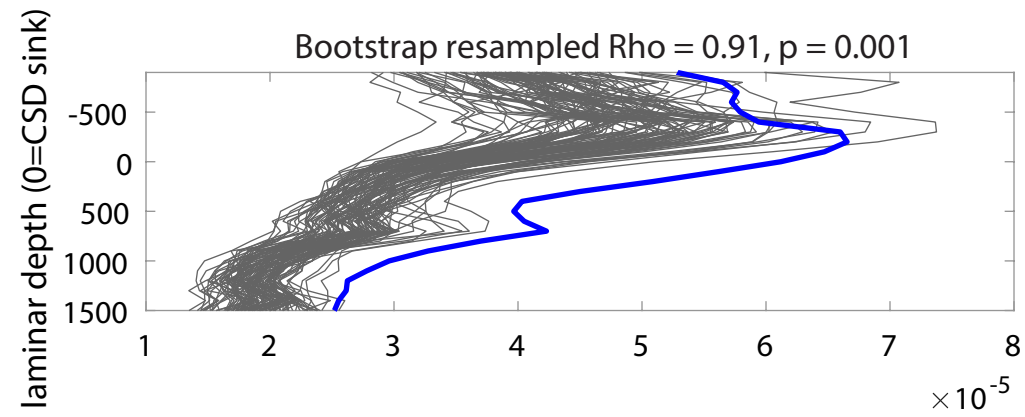


Supplemental Figure 7

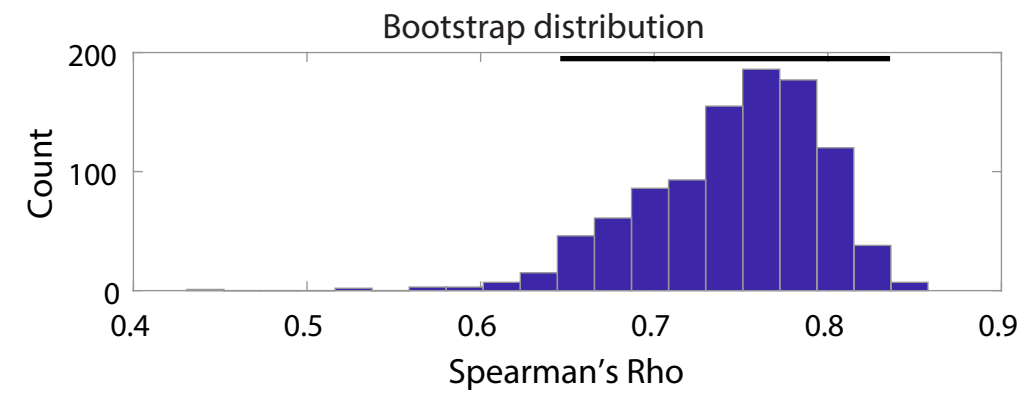
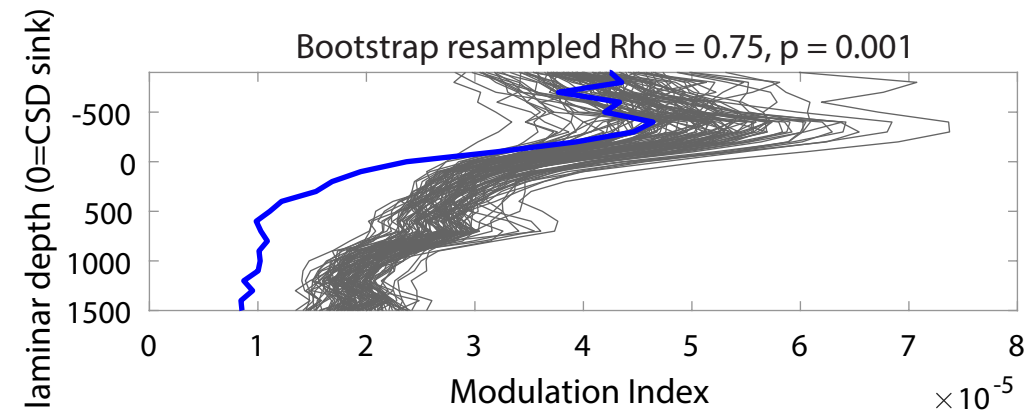


Supplemental Figure 8

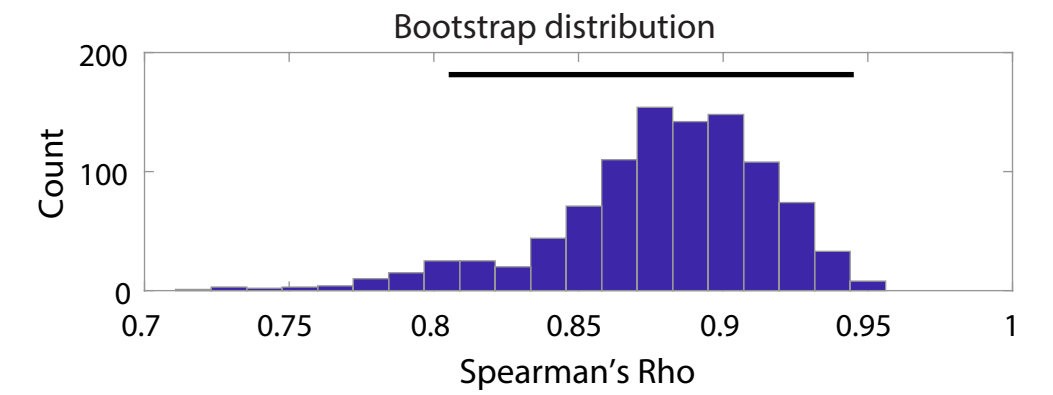
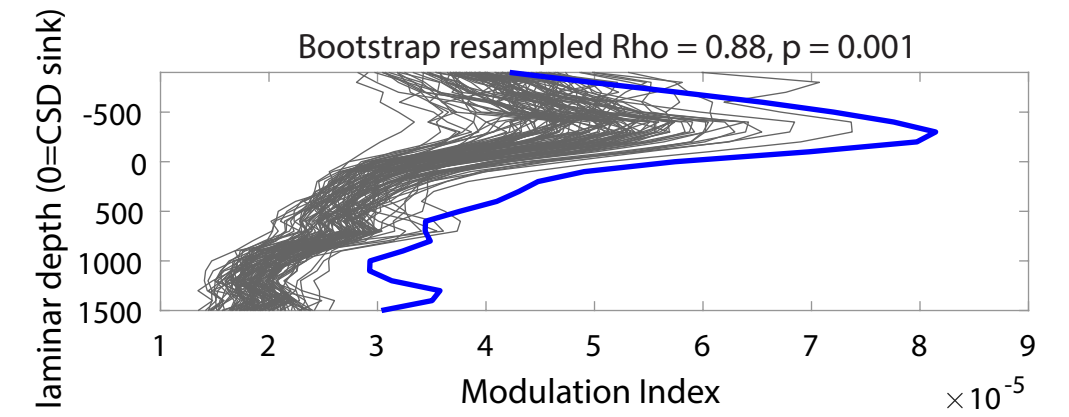
### VLPFC (N=23)



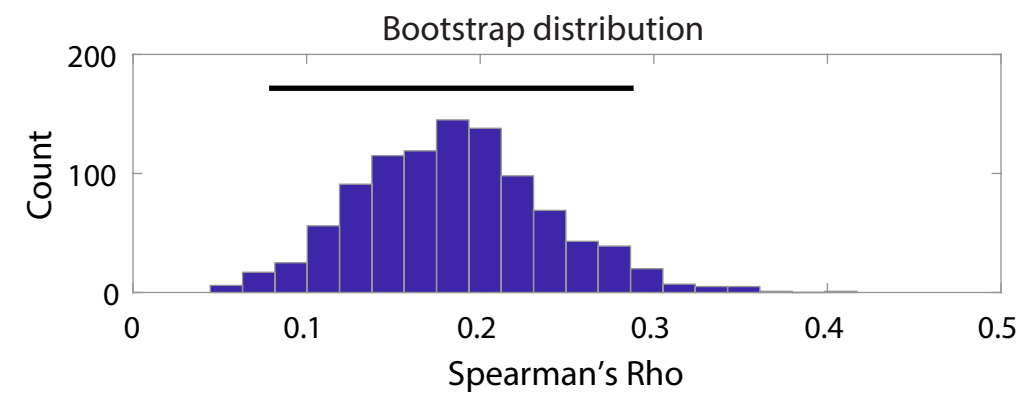
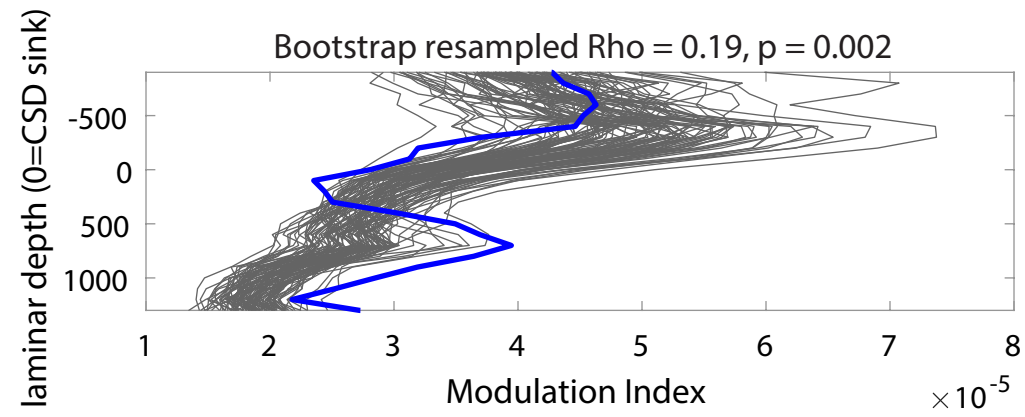
### PMd (N=20)



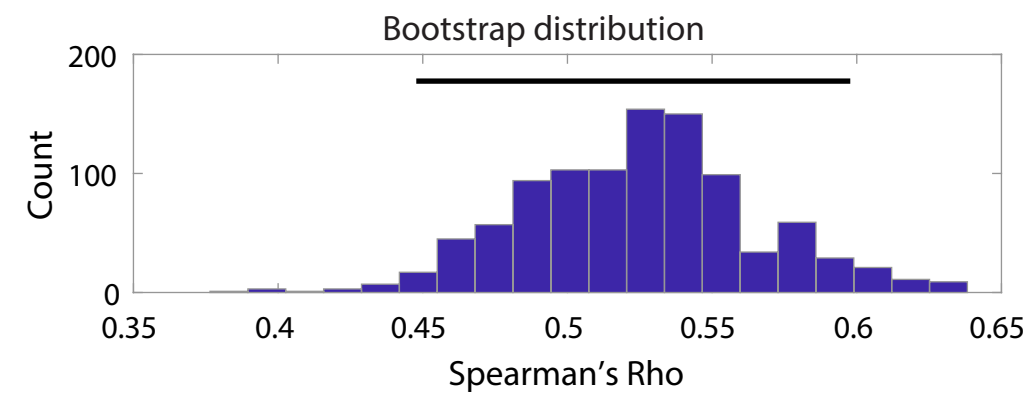
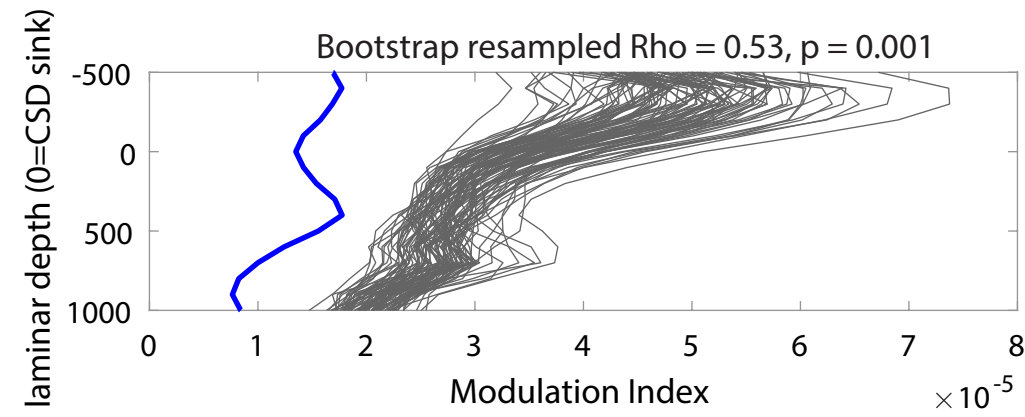
### 8A (N=8)



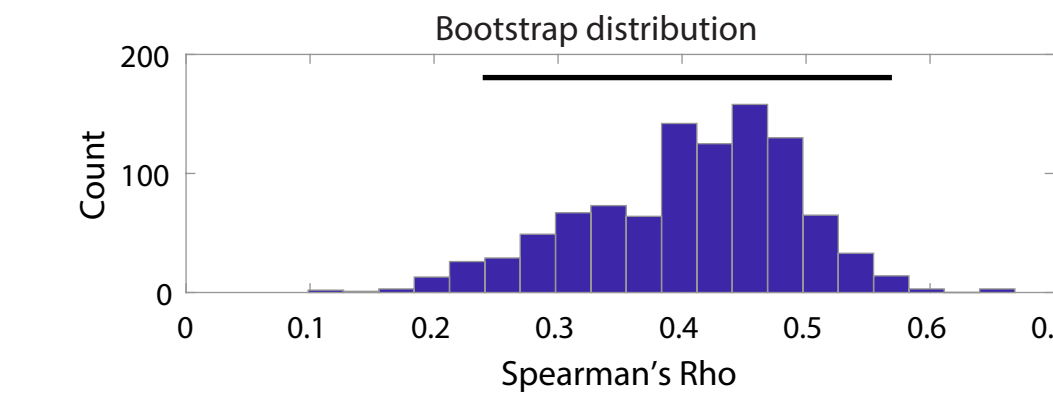
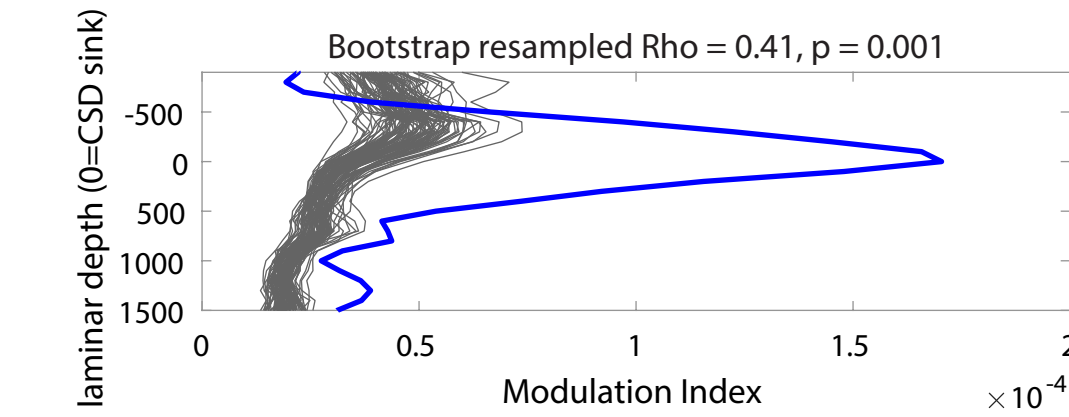
### ACC/SMA (N=4)



### 8B (N=3)

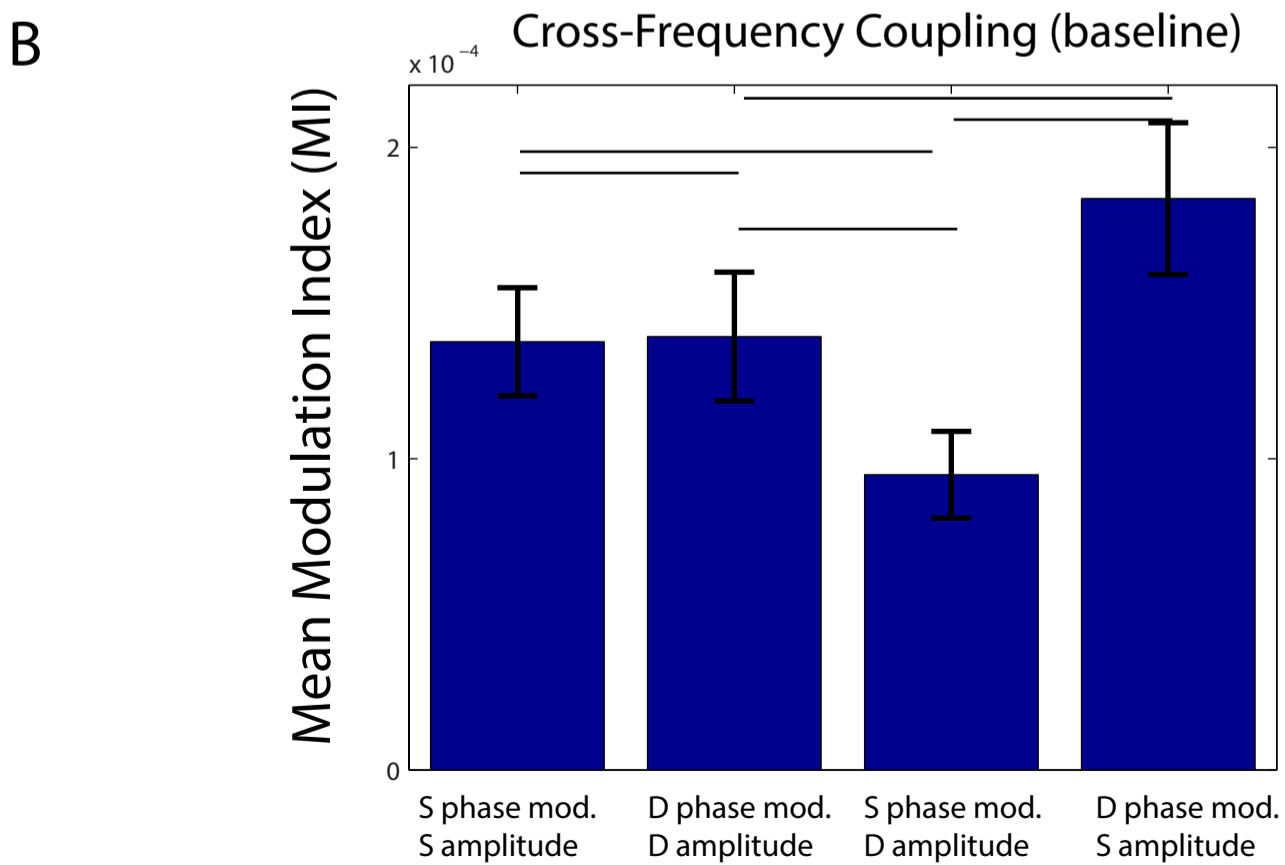
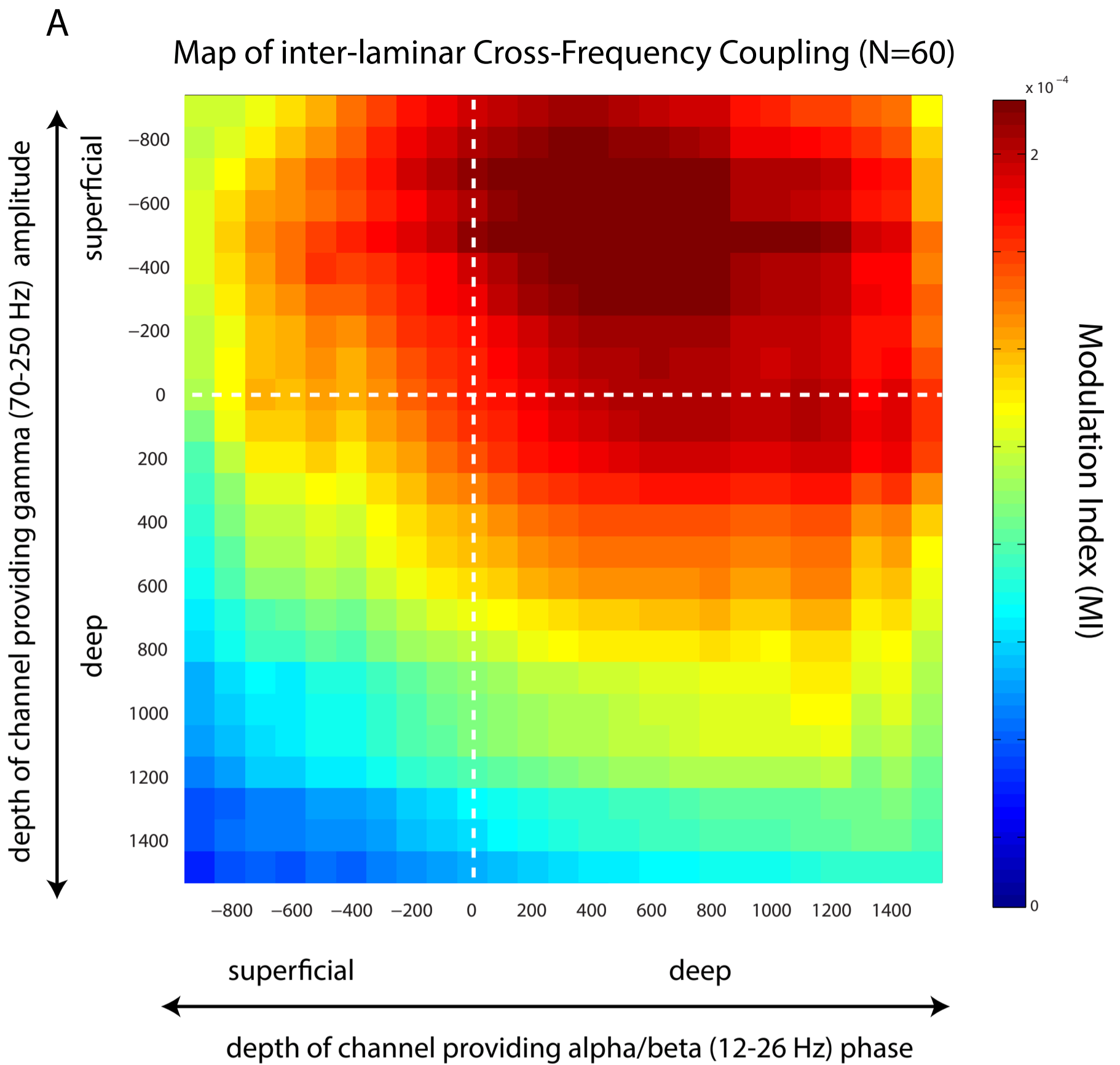


### DLPFC (N=2)



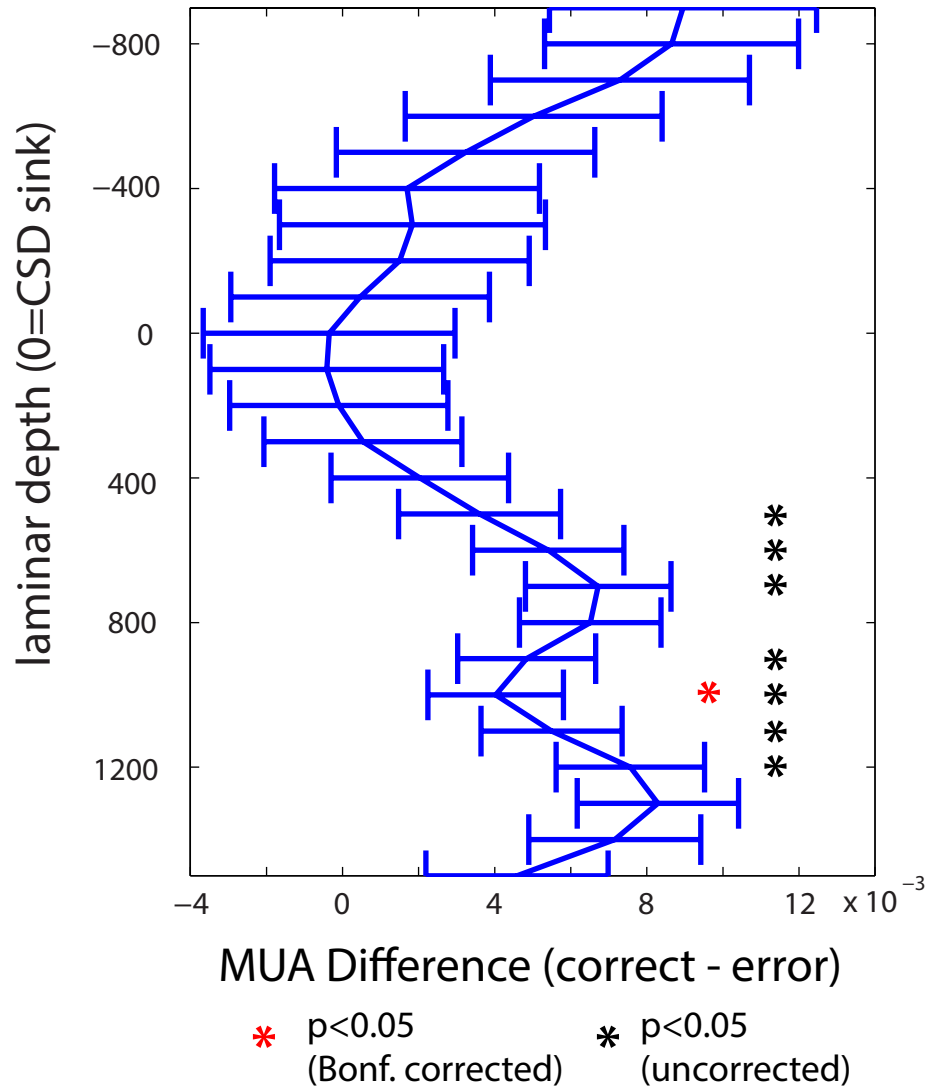
#### Legend

- Cross-Frequency Coupling (resampled average of all other areas)
- Cross-Frequency Coupling (average of each area)
- 95% Confidence Interval for Spearman's Rho

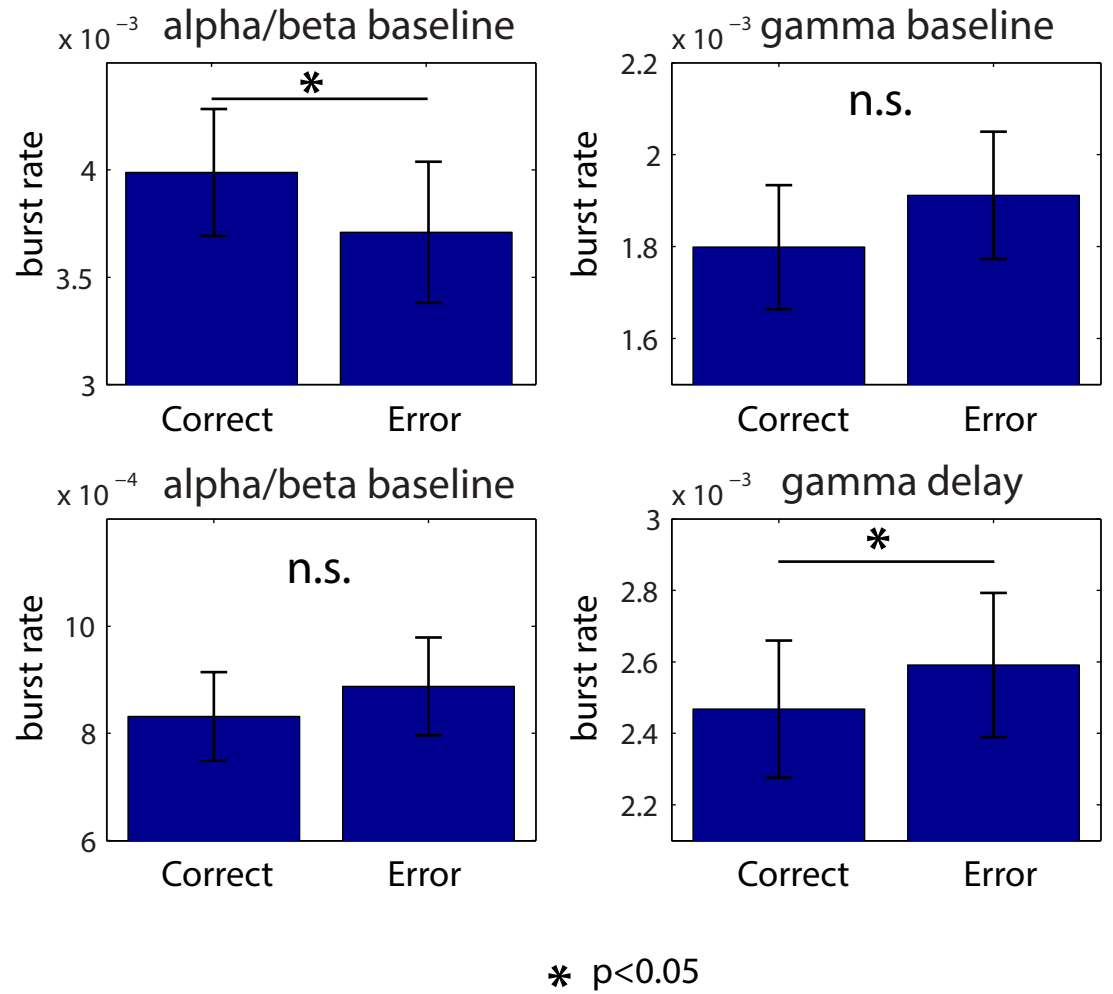


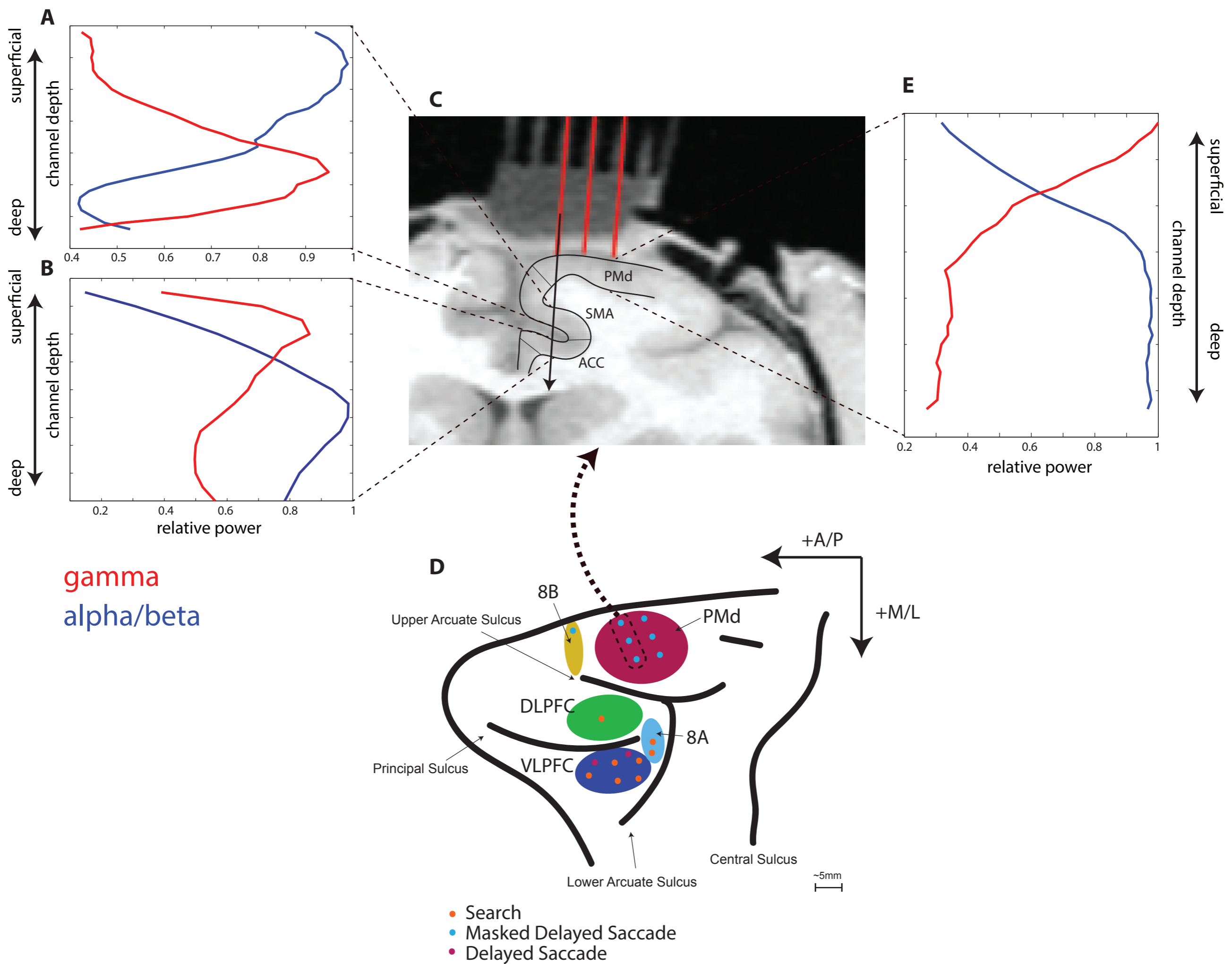
Supplemental Figure 10

**A** Delay-period MUA correct vs. error



**B** LFP bursting correct vs. error





Supplemental Figure 12