Global network influences on local functional connectivity

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A central neuroscientific pursuit is understanding neuronal interactions that support computations underlying cognition and behavior. Although neurons interact across disparate scales, from cortical columns to whole-brain networks, research has been restricted to one scale at a time. We measured local interactions through multi-neuronal recordings while accessing global networks using scalp electroencephalography (EEG) in rhesus macaques. We measured spike count correlation, an index of functional connectivity with computational relevance, and EEG oscillations, which have been linked to various cognitive functions. We found a non-monotonic relationship between EEG oscillation amplitude and spike count correlation, contrary to the intuitive expectation of a direct relationship. With a widely used network model, we replicated these findings by incorporating a private signal targeting inhibitory neurons, a common mechanism proposed for gain modulation. Finally, we found that spike count correlation explained nonlinearities in the relationship between EEG oscillations and response time in a spatial selective attention task.

Action potentials, or spikes, are widely held to be the computational currency of the brain. Decades of research have identified numerous ways in which the activity of individual neurons is related to stimuli in the outside world and to our perception of those stimuli. Cognitive and perceptual processes, however, are not the product of any individual neuron’s activity, but are instead network-level phenomena in which groups of neurons act in concert. These phenomena can be studied by local recording of many neurons simultaneously, via a multielectrode array or imaging of a voltage-sensitive dye, or through more global measures of neuronal activity, such as functional magnetic resonance imaging (fMRI) or EEG. A complete understanding of the neural basis of perception and behavior requires a bridge across these levels of analysis.

Investigations of small populations of neurons have focused on pairwise interactions such as correlated variability in firing rates from trial to trial, also known as spike count correlation (or noise correlation, r_{nc}), a measure of functional connectivity with known implications for coding. Several recent investigations of spike count correlation have found that it is highly structured and modulated by cognitive and perceptual context. However, identification of the signals that generate these dynamics has proved elusive, instead relying on speculation about the large-scale networks involved. Previous attempts to measure the interdependence of activity between brain areas have made tantalizing suggestions that spiking activity can be related to oscillations supported by large-scale networks, but, generally speaking, such networks have proved inaccessible to microscale methods.

The most widely used methods for measuring large-scale network activity are fMRI and EEG. With these methods, an explanatory gap persists as to how the large-scale signals are related to the spiking activity of small populations of neurons. A fair amount of investigation has been directed at linking spiking activity to the fMRI blood oxygenation level-dependent (BOLD) response, but far less research has sought to relate spiking activity and EEG. The EEG is thought to reflect the postsynaptic potentials in the apical dendrites of pyramidal cells resulting from their mutual alignment, which allows summation of electric fields. The strength of the signal is related to both the magnitude of the postsynaptic activity and its coherence: postsynaptic currents with low spatio-temporal coherence tend to destructively interfere at the level of the scalp. The common postsynaptic activity that drives variability in the EEG signal likely also generates spike count correlation across neurons.

We sought to test whether EEG oscillations index the coordination of the spiking activity of the underlying neuronal population by using simultaneous recordings of evoked and spontaneous activity at the scalp and in the cortex of behaving macaque monkeys. We found that oscillations at the level of the EEG did, in fact, relate to spike count correlation, but they did so in a non-monotonic fashion. However, we found that a variation of a widely used simple network model incorporating excitatory and inhibitory subpopulations could account for this relationship. Finally, we report that knowledge of the non-monotonic relationship between EEG oscillations and spike count correlation can explain the connection between EEG oscillations and performance on a spatial selective attention task.

RESULTS

We simultaneously recorded EEG from the scalp along with spiking activity from a ‘Utah’ microelectrode array implanted in area V4 of two macaque monkeys performing a fixation task. We started by isolating a snippet of EEG around the time of each recorded spike.
We then Fourier-transformed those EEG snippets to determine the phase and amplitude of the global oscillations relative to the local spiking activity. To measure spike count correlation among neurons, we created surrogate spike trains (subsets of the full recording) in which the amplitude and phase of the EEG were in a specified range. This allowed us to ascertain how spike count correlation related to particular properties in the EEG. A simplified understanding of our approach can be visualized by thresholding the envelope of the continuously filtered EEG signal and apportioning spikes accordingly (Fig. 1b). On the full data set, our actual method employed the spike-triggered Fourier coefficients to create surrogate spike trains (Online Methods).

Relationship between EEG and spike count correlation
Surrogate data from a representative session illustrate one of the more robust and surprising effects that we observed (Fig. 2). We predicted that spike count correlation would be directly related to oscillation amplitude, based on the assumption that greater amplitude oscillations are a result of more coherent input to the underlying brain area. In contrast with this prediction, we found that spiking activity during the intermediate amplitudes of alpha oscillations not only had relatively less spike count correlation than spiking activity during higher amplitude oscillations (which we predicted), but also had less spike count correlation than spiking activity during lower amplitude oscillations. In other words, the magnitude of spike count correlation followed a U-shaped relationship with EEG oscillation amplitude. Although the overall magnitude of spike count correlation varied somewhat from session to session, the U-shaped relationship between oscillation amplitude and spike count correlation was highly reliable (Fig. 3a). This relationship was statistically significant (P = 0.01), as determined by a permutation test consisting of randomly shuffled data subjected to the identical analysis procedure (Fig. 3b and Online Methods).

In addition to the U-shaped relationship between EEG oscillation amplitude and spike count correlation, we also found a sinusoidal relationship between EEG oscillation phase and spike count correlation in the ‘spontaneous’ activity condition (Fig. 3a). This effect was seen for all frequencies. Changes in correlation state with the
Our initial analysis focused on alpha oscillations because of their historical prominence in the EEG literature, but we found that the U-shaped relationship between EEG oscillation amplitude and spike count correlation was evident across frequencies. Each of the frequency bands of interest revealed a U-shaped relationship between amplitude and correlation, but the strength of this relationship was directly related to frequency: low-frequency oscillations showed relatively less modulation of spike count correlation with amplitude and high-frequency oscillations showed relatively greater modulation of correlation with amplitude. The magnitude of the relationship between EEG oscillation amplitude and spike count correlation also displayed spatial specificity, with the strongest relationships being seen for EEG measured at the electrode nearest the array and at the electrode diametrically across skull. This spatial specificity was most evident for the frequencies with the greatest effect sizes. The antipodal topographical pattern that we observed is generally consistent with both the positive pole and negative pole of a single dipole source located near the array reflecting the amplitude-correlation relationship.

All of these analyses were performed on the basis of spontaneous spiking and EEG activity recorded during fixation. When a stimulus was presented in the receptive field of the neurons recorded on the array, we saw a similar U-shaped relationship between EEG oscillation amplitude and spike count correlation to what we found for the spontaneous condition. However, we did not observe a relationship between correlation and EEG phase in the evoked condition, indicating that the visual stimulus abolished the phase-locking between the spikes and the EEG. Analysis of the firing rate of our pairs of neurons was more complicated due to the presence of stimulus-related modulations in firing rate.
value in relating EEG (the predominant electrophysiological method in humans) to spiking activity, an important question is whether the same mechanisms operate at the scale of hundreds of micrometers (LFP) as at the scale of centimeters (EEG). We therefore performed the same analysis on the stimulus-evoked condition using the LFP from a randomly chosen electrode in the array (3 of 18 data sets were excluded because of errors saving the LFP data). We found that the LFP-\( r_{SC} \) relationship closely mirrored the EEG-\( r_{SC} \) relationship both qualitatively and quantitatively. To assess this, we measured the Pearson’s correlation between the data points comprising the two sets of curves and found significant positive correlations at each frequency (all \( r \) values > 0.72, all \( P \) values < 0.001, one-sample \( t \) test). The magnitude of the modulation (max – min) of \( r_{SC} \) did not differ significantly whether based on oscillations in the EEG or the LFP (\( P = 0.24 \) across all frequencies, paired-samples \( t \) test). These results are consistent with the EEG and LFP both reflecting the same mechanisms that modulate spiking activity.

Potential role of cross-frequency interactions in EEG

We were surprised to find a U-shaped relationship of spike count correlation to oscillation amplitude. Our guiding prediction had rather been that spike count correlation would increase monotonically with oscillation amplitude, under the reasoning that greater amplitude oscillations would be generated by more coherent postsynaptic currents in the underlying brain area, and that more coherent postsynaptic currents would lead to correlated spike output. This reasoning can account for the pattern of results that we observed at high amplitudes, where amplitude and spike count correlation were directly

**Figure 6** Results for evoked task. The results illustrated are for alpha-band EEG measured at the right occipital electrode, but similar results were seen across frequencies and at other electrodes. (a) Correlation results. Data are presented as in Figure 3a. (b) Firing rate results. Data are presented as in Figure 5. Note that phase effects were greatly reduced for the evoked task compared with the spontaneous task. Error bars represent ± s.e.m.
related, but it cannot explain the pattern of results that we observed at low amplitudes, where amplitude and spike count correlation were inversely related.

Given that cross-frequency EEG interactions have been linked to multiunit activity, we wondered whether dependencies between the frequency bands might lead to the non-monotonic effect that we observed. If the amplitudes of some of our frequency bands tended to be inversely related to each other, then this could potentially account for the non-monotonicity while remaining consistent with the essentially monotonic mechanism that guided our initial predictions. In other words, two oscillations that consistently trade-off in amplitude from trial to trial could drive changes in spike count correlation, giving merely the appearance of a non-monotonic mechanism. This was not the case, however, as we calculated the Pearson correlation of amplitude between all pairings of the six frequencies of interest and found that no pair of frequencies was inversely related. We did find significant positive correlations between some frequencies, however. In particular, lower frequencies (delta, theta and alpha) were correlated with each other in the range of \( r = 0.02–0.08 \), and higher frequencies (alpha, beta, low gamma and high gamma) were correlated with each other in the range of \( r = 0.08–0.26 \). The highest frequency pairs had the greatest correlation, which is consistent with the diminishing frequency resolution achieved as frequency increases. In general, it is not surprising that nearby frequencies were correlated, simply as a result of their proximity. The only significant correlation that we observed between distant frequency bands was between delta and low gamma \( (r = 0.05, P = 0.002) \), which is noteworthy because interactions between these two frequency bands in particular has recently been linked to multiunit activity. To summarize, our finding that none of the pairs of frequencies was inversely correlated rules out amplitude trade-offs between frequency bands as a potential cause of the non-monotonic amplitude-correlation relationship.

Neural network modeling

We next asked whether a network of interconnected neurons could produce the non-monotonic relationship by virtue of the complexity of the dynamics that a neural network can generate. We modeled a balanced network of excitatory and inhibitory neurons driven by an external population of random, doubly Poisson neurons and an additional applied current. The parameters for the model were set based on previous literature and we did not modify them; rather, we only modified the input currents that we applied to this ‘off-the-shelf’ model. We used band-filtered (alpha band, 8 – 12 Hz) white-noise signals for our applied current to model a coherent oscillatory input to the network. We modeled the resulting spike train for each neuron in the network and we modeled the corresponding EEG as the summed postsynaptic potential of the excitatory neurons in the network. We divided our set of modeled trials into decile bins by EEG alpha amplitude and calculated the average spike count correlation in each bin.

When we applied a single oscillatory current ranging from 0–0.3 nA in amplitude identically to all the neurons in the network, we found that correlation between model excitatory neurons was on the order of \( 1/N (r_{EE} = 0.001; \text{Fig. 7a}) \), consistent with prior calculations demonstrating the ability of a balanced network to cancel input correlations. When much larger amplitude oscillation amplitudes were included (>0.3 nA), correlation increased monotonically with alpha amplitude as the spiking activity became unnaturally coherent with the driving oscillation (data not shown). Because the spiking behavior induced by these large-amplitude input oscillations was physiologically implausible, we restricted our subsequent analyses to input oscillations below that range. To summarize, the simple balanced neural network with a single input shared by the entire network could not reproduce the non-monotonic behavior that we observed in our experiment.

We next tested whether applying separate input oscillations to the excitatory and inhibitory subpopulations of our network might produce a non-monotonic relationship between EEG amplitude and correlation. We were guided in this hypothesis by the observation that feedback signals tend to target these two cell types differentially. For example, in V4, attention-dependent modulation of the activity of putative inhibitory neurons is stronger than that for putative excitatory neurons (using waveform shape to separate neurons into subclasses). When we applied a separate input oscillation to the inhibitory subpopulation (0–0.2 nA), in addition to the global input oscillation that was shared by all the neurons, this resulted in a U-shaped relationship between EEG alpha amplitude and spike count correlation that matched our experimental data (Fig. 7a). This effect was robust over the entire range of input amplitudes of the model that we tested that produced physiologically plausible spike trains (Fig. 7b). This indicates that a balanced neural network is sufficient to generate the non-monotonic relationship that we found if the inhibitory subpopulation receives a separate input oscillation in addition to that shared with the population at large.

It is notable that our neural network model is spatially homogeneous—that is, neurons are connected to each other randomly without regard for their relative spatial positions. Functional connections among real neurons are known to decay with distance and tuning similarity, and, in our own data, we saw that the relationship between EEG and spike count correlation was strongest for scalp locations closest to the multielectrode array. Extensions of this model will need to incorporate these distance effects as well as tissue conduction to more fully model the EEG. An additional important question is how critical it was that we applied band-limited input to our neural network model, as such input might not be ecologically typical (although intrinsic filtering properties of dendritic membranes may make individual synapses particularly sensitive to limited frequency bands). Two observations suggest that band-limited input is not critical. First, although our input oscillation was band-limited to the

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Figure 7 Neural network modeling results. (a) Spike count correlation between excitatory neurons \( (r_{EE}) \) was on the order of \( 1/N \) if global input oscillations were only applied to the model (gray) and did not vary substantially with modeled EEG alpha power. If an independent input oscillation was applied to the inhibitory population, the resulting \( r_{EE} \) spanned a range similar to our observed results and followed a U-shaped profile as a function of modeled EEG alpha power (black). (b) Amplitude-correlation relationship as a function of global oscillation amplitude. The U-shaped amplitude-correlation relationship was robust when considering trials derived using a single global input oscillation (and a mixture of private inhibitory input oscillations) across a broad range of global input oscillation magnitudes.
alpha range, the resulting modeled EEG had a broad-band power spectrum by virtue of the internal network dynamics (Supplementary Fig. 1). Second, we also observed a U-shaped relationship between correlation and EEG amplitude at all other resolvable frequency bands in the model (delta was below our resolution) that were qualitatively similar to what we observed in vivo. Because both the field potentials and the effects on correlation generated by our neural network model were not limited to the input frequency band, this neural network model is consistent with the essentially broad-band field potentials that have been previously linked to spiking activity in vivo26,33.

Spatial attention task
In a variety of contexts, psychophysical performance has been found to follow a U-shaped relationship with EEG oscillation power34–37, much like the relationship we found between the EEG and spike count correlation. To date, interpretations of this observation have been speculative, typically invoking the notion of stochastic resonance. Our results, however, hint at an alternative explanation: changes in spike count correlation could serve as the computational basis for this effect, as decorrelation has been previously linked to improved performance in an attention task2, potentially as a result of improved signal averaging during the decorrelated state1. We therefore sought to test this hypothesis by performing an additional experiment.

Two animals were trained on a selective attention task in which they maintained central fixation while two drifting gratings were presented; one in the receptive field area and one in the mirror symmetric position in the opposite hemifield. The task was to detect a change in the drift speed of one of the two gratings and to make an eye movement to the grating, if any, that changed, or else to maintain fixation until the end of the trial (1.2 s). On most trials, a briefly flashed visual cue indicated the stimulus location that was most likely to contain the target, and analysis of behavioral data indicated that this cue resulted in a robust enhancement of performance at the cued location.

For the analysis of this experiment, we focused on the amplitude in the alpha frequency band of the EEG, which has been most consistently linked to selective attention26. Our goal was to determine whether the underlying spike count correlation mediates a U-shaped relationship between EEG alpha amplitude and response time. We divided trials into six bins based on alpha amplitude and calculated spike count correlation over the trials in each bin. Following a previously described procedure38, we used a regression analysis to determine the degree to which alpha band amplitude could explain variance in behavioral response time to targets contralateral to the array, and the degree to which this relationship was mediated by spike count correlation. We found that response time indeed followed a U-shaped relationship with alpha amplitude measured at the parieto-occipital electrode over the array, with the fastest response times observed at intermediate alpha amplitudes (Fig. 8). Inclusion of a linear term for correlation improved the variance accounted for by the regression equation (by 118% for Monkey B, \( P = 0.011 \); 240% for Monkey R, \( P = 0.042 \)). Notably for our predictions, we found that inclusion of spike count correlation in the regression equation led to a decrease of the quadratic term for alpha amplitude (by 15% for Monkey B, \( P = 0.047 \); 16% for Monkey R, \( P = 0.030 \)). In other words, the U-shaped relationship between response time and alpha amplitude was substantially mediated by the degree of spike count correlation in the underlying area.

To illustrate the effect of this mediation, we used the resultant regression equation to model the predicted response times for each alpha amplitude while adjusting for the effect of spike count correlation, which resulted in a marked flattening of the relationship (Fig. 8). This relationship with behavior was specific to the alpha band (for all other frequency bands for both subjects; all \( P \) values > 0.186).

DISCUSSION
We found that functional connectivity among spiking neurons (measured by spike count correlation) followed a U-shaped relationship with EEG spectral power measured at the scalp. This result is counter to the basic intuition that increasing EEG power would reflect increasingly coherent input to the underlying neurons and, in turn, lead to an increase in spike count correlation. The relationship that we found was topographically specific, with the EEG electrode sites nearest the multi-electrode array showing the strongest effects, but with additional contributions from the diagnostically opposing sites, consistent with the two poles of a dipole source. We also found the general pattern to be robust across frequencies, although higher frequencies had stronger amplitude-correlation relationships. Our results provide a powerful insight into the relationship between local neuronal populations and global brain activity, and advance our understanding of how the functional connectivity of small scale networks is modulated by the context of global brain states, which is a fundamental aspect of brain function.

To further understand our results, we employed a popular computational model using a balanced network of excitatory and inhibitory neurons17,18. In its basic form, this model effectively cancels correlations in common input and produces an asynchronous network of neurons with near-zero mean correlations. We first considered the simplest modification to this model, a global excitatory oscillatory input applied to all of the neurons, which failed to produce the...
behavior that we observed in vivo and instead produced a monotonic increase in correlations with excitatory oscillations of sufficient magnitude. We then considered a second addition to the model, in which we delivered an independent oscillation exclusively to the inhibitory subpopulation of neurons. Although the spatially homogeneous organization of dendrites in inhibitory interneurons would largely hide such an oscillation at the level of the EEG, it produced a non-monotonic relationship between EEG power and spike count correlation that was an excellent match to the physiological data. That a relatively simple and well-studied model reproduces our in vivo findings while implicating a specific subpopulation of neurons provides a powerful link to phenomena proposed to depend on selective activation of inhibition, such as attention, sensory binding and memory formation.

For example, several recent studies have reported an inverted-U effect of EEG oscillations during attention-demanding detection tasks, with performance varying non-monotonically with EEG power. Without concurrent population recordings, such studies are left to speculate high-level functional explanations of behavior, invoking phenomena such as stochastic resonance. Our data confirm that the inverted U shape relating EEG oscillations to performance during attention in these studies can be explained by the decorrelation in spiking observed at intermediate oscillation amplitudes, which reflects a change in processing state of the neuronal population. Moreover, our model posits a key role for inputs to inhibitory neurons underlying this non-monotonic effect. Indeed, modulation of inhibitory subpopulations by long-range feedback signals appears to be a key mechanism to adjust gain during selective attention and may be mediated by cholinergic receptors on inhibitory neurons. Our observations thus connect studies of attention in humans, in vivo brain networks. These findings highlight the critical role of observations across scale in revealing the mechanisms by which neuronal networks give rise to complex perceptual and cognitive experiences.

**METHODS**

Methods and any associated references are available in the online version of the paper.

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**AUTHOR CONTRIBUTIONS**

A.C.S. and M.A.S. designed the experiments, analyzed the data and wrote the manuscript. A.C.S. and C.M.W. conducted the experiments. A.C.S. and M.J.M. conducted the network model simulations. M.A.S. supervised the project.

**COMPETING FINANCIAL INTERESTS**

The authors declare no competing financial interests.

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We recorded EEG from 8 Ag/AgCl electrodes (Grass) in V4 in each of three adult male rhesus macaques (Macaca mulatta). We implanted the arrays in the right hemisphere for Monkeys W and B, and in the left hemisphere for Monkey R. The basic surgical procedures have been described previously4 and were conducted in aseptic conditions under isoflurane anesthesia. In addition to the microelectrode arrays, the animals were implanted with a titanium head post to immobilize the head during experiments. We recorded neurons with receptive fields centered 3.16°, 5.66° or 10.23° from the fovea in the lower visual field of each animal. Experimental procedures were approved by the Institutional Animal Care and Use Committee of the University of Pittsburgh.

Behavioral tasks. We presented visual stimuli with custom software written in MATLAB (MathWorks) using the Psychophysics Toolbox extensions47–49. Monkeys W and B performed the following tasks. We trained the subjects to maintain fixation on a 0.6° blue dot at the center of a flat-screen cathode ray tube monitor positioned 36 cm from their eyes. The background of the display was 50% gray. We measured the monitor luminance gamma functions using a photometer and linearized the relationship between input voltage and output luminance using lookup tables. In the spontaneous task, subjects were trained to maintain fixation on the central dot for 2 s, at which time the fixation point would be moved 11.6° in a random direction and the animal received a liquid reinforcement for making a saccade to the new location. No other stimuli were presented for the spontaneous task. The evoked task was identical, except that we presented a drifting sinusoidal grating (100% contrast, maximum luminance of 145 cd m−2) in the aggregate receptive field (RF) area of the neurons recorded on the microelectrode arrays during the 2-s fixation interval. Both subjects completed the spontaneous task (Monkey B, 14 sessions; Monkey W, 10 sessions) before performing the evoked task (Monkey B, 10 sessions; Monkey W, 8 sessions).

Microelectrode array recordings. Signals from the microelectrode arrays were band-pass filtered (0.3–7500 Hz), digitized at 30 kHz and amplified by a Grapevine system (Ripple). Signals crossing a threshold (periodically adjusted using a multiple of the r.m.s. noise for each channel) were stored for offline analysis. These waveform segments were sorted using an automated clustering algorithm30 followed by manual refinement using custom MATLAB software52 (available at http://www.smithlab.net/spikesort.html), taking into account the waveform shapes and interspike interval distributions. After sorting, we calculated the signal-to-noise (SNR) ratio of each candidate unit as the ratio of the average waveform amplitude to the s.d. of the waveform noise.52 Candidates with an SNR below 2.5 were discarded.

RF mapping and tuning curves. Prior to beginning the experiments, we mapped the RF areas of the units recorded on our arrays by presenting small (~1°) sinusoidal gratings at four orientations positioned one at a time on the vertices of a lattice in the likely RF area per the anatomical location of the implant. After inspecting the responses of the units to these small probe stimuli, we picked a stimulus size and position to roughly cover the aggregate RF area. For Monkey B this was 5.87° diameter centered 4.00° below and 4.00° to the left of fixation, for Monkey W this was 4.70° diameter centered 1.18° below and 2.94° to the left of fixation, and for Monkey R this was 5.87° diameter centered 8.94° below and 4.99° to the right of fixation. Throughout this report, we use RF to refer to this aggregate area. We next measured tuning curves for the recorded units by presenting sinusoidal gratings to the RF area in four orientations and at a variety of spatial and temporal frequencies. For each subject we chose a compromise temporal and spatial frequency that evoked the maximum response from the array as a whole.

EEG recordings. We recorded EEG from 8 Ag/AgCl electrodes (Grass Technologies) adhered to the scalp with conductive paste (see Fig. 1a for positioning). Signals were referenced online to the head post, digitized at 1 kHz and amplified by a Grapevine system (Ripple) and low-pass filtered online at 250 Hz. Data were first divided into trial epochs time-locked to the onset of the grating stimuli (~0.5–3 s); analyses focused on subsets of this interval. Trials containing excessive muscle activity or transient artifacts were rejected using an automatic threshold criterion (~300 μV) and visual inspection. After artifact rejection, data were re-referenced to the average activity across all electrodes.

Eye tracking. We tracked the gaze of the subjects using an infrared eye tracking system (EyeLink 1000, SR Research). Gaze was monitored online by the experimental control software to ensure that subjects maintained fixation within 1.17° of the central fixation point during each trial.

Data analysis. To examine the relationship between spectral properties of the EEG and the spike count correlation of the neurons recorded on our microelectrode arrays, we constructed surrogate spike trains characterized by particular phase and amplitude properties (similar to a previously described procedure27). Specifically, we focused on the six frequency bands analyzed in a previous study16, which compared spiking activity to a single EEG ring electrode: 2–4 Hz (delta), 4–8 Hz (theta), 8–12 Hz (alpha), 12–30 Hz (beta), 30–60 Hz (low gamma) and 60–100 Hz (high gamma). Average power spectra for Monkeys B and W, measured over the 2-s fixation periods, are illustrated in Supplementary Figure 2. For each spike, we calculated the fast Fourier transform of the 500-ms Hanning-windowed segment of each channel of EEG data centered at the time of the spike. Note that spikes occurring in the first or final 250 ms of a trial were therefore excluded because the window would have overlapped the trial boundary. These spike-triggered spectra were used to compute the average amplitude across each frequency band around the time of each spike, as well as the phase of the center frequency for each frequency band around the time of each spike. We next created virtual spike trains for each combination of 10 amplitude ranges and 12 phase ranges measured at each EEG channel and for each frequency band. We divided amplitude measurements into 10 deciles that each contained an equal number of spikes. We divided phase into 12 bins, each subtending 60 degrees of phase, with 50% overlap between adjacent bins. In a separate control analysis, we used six, non-overlapping phase bins spaced to equate as closely as possible the number of spikes in each bin for each session. Each virtual spike train spanned 1.5 s of real time, but consisted only of spikes that occurred when the amplitude and phase of a given frequency band measured at a given electrode was within the specified ranges. We summed the spikes of these surrogate spike trains to produce a single spike count for each 2-s trial. We normalized the spike counts of each neuron for each grating orientation by z-scoring. We then computed the spike count correlation (ρsc) as the Pearson product moment correlation coefficient of normalized spike counts for all pairs of neurons recorded simultaneously from separate microelectrodes. Correlation was measured separately for each recording session.

Statistical analysis. We used curve-fitting combined with a non-parametric resampling procedure to quantify our statistical confidence in the relationships between spectral properties of the EEG and spike count correlation. We first fit curves (described below) to our observed data in the ordinary least-squares sense, and quantified the strength of the relationship according to the fitted function. We then randomly shuffled the pairings between spikes and EEG data windows, so that the relationship between spikes and EEG was random, which represents the null hypothesis. We repeated the analysis making surrogate spike trains for specific EEG spectral properties, this time using the randomly-shuffled data. We then fit curves to the results from the shuffled-data analysis. The data shuffling and reanalysis were iterated a total of 1,000 times to create a distribution of resultant curve fits under the null hypothesis. The proportion of this distribution that exceeded the observed value was the p-value for the claim that the relationship we observed was stronger than would be expected by chance. We considered P < 0.05 to be significant. All statistics were performed across sessions, treating the data from each day as a unit of observation. We viewed this to be the most conservative approach, when compared with the alternative of adding together the pairs across days and treating each pair as an observation.

To characterize the U-shaped relationship between EEG oscillation amplitude and spike count correlation, the function we fit to the data was a ratio of two quadratics. This is the simplest function that can be non-monotonic and can also have a bounded range (since correlation must be bounded at least between −1 and 1). We collapsed across phase for this fit, and considered only amplitude. We measured the coefficient of determination, adjusted for the number of free parameters (that is, R2), for both the rational function fit, as well as for a simple linear fit. We quantified the strength of the non-monotonic relationship as the change in adjusted coefficient of determination from the linear fit to the rational fit (ΔR2).
To test whether the presence of small eye movements could have confounded our results, we performed a median split of trials for each experimental session based on gaze position variance, and repeated the analysis described above. Since gaze position is two-dimensional, we derived a scalar measure of gaze position variance using the generalized variance

$$
\Sigma = \sqrt{\text{det}(XX^T)}
$$

where $X$ is a $n \times 2$ matrix of gaze position coordinates, and $n$ is the number of samples in the 2-s analysis window. After we repeated our analysis on both halves of trials for each session, we tested for differences between trials with relatively stable gaze positions and trials with relatively variable gaze positions using independent samples t tests at each amplitude of each frequency for both conditions (a total of 120 tests). We did not adjust the reported p-values for the family-wise error rate.

To test for amplitude covariance between the frequency bands of interest, we measured the amplitude of each of the six frequency bands of interest by windowing each 2-s trial with a Hann function and then applying the fast Fourier transform. We calculated the Pearson product-moment correlation coefficient of amplitude over trials for each pair of frequencies for each recording session (spontaneous and evoked conditions). We applied Fisher's r-to-z transformation to the correlation values, and then tested the distributions of transformed values against the null hypothesis of zero correlation with a two-tailed Student's t test at $\alpha = 0.05$, Bonferroni-corrected for the 15 pairs of frequency bands.

**Neural network modeling.** We simulated spiking neuron populations using a conductance-based leaky integrate-and-fire network with band-pass (alpha band, 8−12 Hz) noise oscillatory input, adapted from a previous study. Briefly, the network was comprised of three populations—one excitatory (E; $N_E = 1,000$ neurons), one inhibitory (I; $N_I = 250$ neurons) and one input (X; $N_X = 1,000$ neurons) population—and was iterated with time step $\Delta t = 0.05$ ms in a Runge-Kutta-2 integration scheme using MATLAB. The membrane voltage of the ith neuron in the $\alpha$ population, $V_i^\alpha$, was governed by

$$
\frac{dV_i^\alpha}{dt} = -g_L(V_i^\alpha - V_L) + I_{\text{syn}}(E) + I_{\text{syn}}(I) + I_{\text{app}}
$$

where $C_m = 0.25$ nF was the membrane conductance, $g_L = 16.7$ nS was the leak conductance (such that the membrane time constant $\tau_m = 15$ ms), $V_L = -70$ mV was the resting potential and $\theta = -50$ mV was the spiking threshold. Following a spike, the voltage $V_i^\alpha$ was reset to $V_L = -60$ mV, after which a refractory period of 2 or 1 ms was assumed for the E and I populations, respectively. All parameters were consistent with the original model.

$\nu_{ij}^\beta$, the synaptic current from population $\beta$ (E, I, or X) onto population $\alpha$ (E or I), was governed by

$$
I_{\text{syn}}(E) = - \sum_{j=1}^{N_E} \rho_{ij}^E g_{ij}^E \alpha_{ij}^E(t) + (V_i^\alpha - V_R^E)
$$

where $V_R^E$ is the reversal current of the efferent population ($V_R^K = V_R^K = 0$ mV and $V_R = -80$ mV). $\rho_{ij}^E$ was the probability that cell i in population $\alpha$ was connected to cell j in population $\beta$, defined for all populations as a binary random variable with a mean of 0.2 (that is, densely but randomly connected). $g_{ij}^E$ was the synaptic conductance from cell i in population $\alpha$ to cell j in population $\beta$, defined as a Gaussian random variable with mean $g_0$ and s.d. 0.5 $g_0$. We used $g_0 = 2.4$ nS, $g_0 = 40$ nS, $g_0 = 4.8$ nS, $g_0 = 40$ nS, and $g_0 = 5.4$ nS. $\alpha_{ij}^E$ was a synaptic gating term, which integrated post-synaptic potentials from the $\beta$ population at the $ij$ synapse according to the following double-exponential ODE (with intermediate variable $\tilde{x}_{ij}^\beta$)

$$
\tau_d \frac{dx_{ij}^\beta}{dt} = -x_{ij}^\beta + \tilde{x}_{ij}^\beta
$$

where $\tau_d$ was spike times of neuron $j$ and $\tau_r = 5$ ms and $\tau_{ab} = 1$ ms were the decay and rise time constants, respectively. $d_{ij}^{ab}$ was the conductance delay between pairs of neurons, defined as a uniform random variable with resolution $\Delta = 0.05$ ms with $d_{ij}^{ab} \in [0.5, 1.5]$ ms and $d_{ab}^{ab} \in [0.1, 0.9]$ ms. The spike times of neurons of the input population (X) were independently Poisson-distributed with mean 10 Hz.

$I_{\text{app}}^\beta$ was an applied oscillatory current to the $\alpha$ population, constructed as white noise that was band-pass-filtered to the alpha band, 8−12 Hz. We used two oscillations: one global oscillation delivered to both E and I populations and a private oscillation delivered only to the I population. To represent varied input oscillation amplitudes, the r.m.s. amplitude of the global and private oscillations were scaled independently; ten different global oscillations ranged from 0−0.60 nA, ten different private oscillations ranged from 0−0.20 nA, and all pairs of amplitudes were simulated. This range was chosen to cover the full range of possible network behaviors under the oscillations. We found that global oscillation amplitudes greater than 0.30 nA yielded ecologically implausible bursting activity coherent with the input oscillation, and therefore focused only on oscillations below that value.

We simulated 48 different networks, each for 6−12 s for each individual pairing of global and private amplitudes. Every segment of data was decomposed into 200-ms trials, giving us frequency resolution at and above 5 Hz. By this method, we accrued at least 1,920 trials for each global and private amplitude pair. We modeled the EEG as the sum of postsynaptic potentials in the excitatory neurons, reasoning that the EEG signal is primarily generated by currents in the apical dendrites of pyramidal cells. We sorted these trials into deciles of the alpha-band amplitude of the modeled EEG (Fig. 7), and pseudo-randomly subsampled the population in each decile bin so that the resulting average pairwise geometric mean firing rate was as nearly constant as possible across bins. Finally, we calculated $r_{ij}$ between pairs of excitatory neurons, which are the predominant cell type recorded on our multielectrode arrays.

**Attention experiment.** Monkeys B and R participated in the attention experiment. The animals were trained to fixate a central yellow dot. After fixating for a randomly chosen duration of 300 or 700 ms, a peripheral visual cue was presented for 120 ms on 89% of trials (no-cue trials were identical in all other respects). For Monkey B, the cue was a 0.6° dim gray dot (87 cd m$^{-2}$, 9% contrast) centered on one of the two locations of subsequently presented grating stimuli. For Monkey R, the cue was a yellow annulus (isoluminant with the display background, 6.45° inner diameter, 7.03° outer diameter, masked to prevent crossing the vertical meridian) that circled one of the two stimulus locations. The cue onset was followed by another randomly-chosen duration of 300 or 700 ms, after which the two drifting sinusoidal gratings were presented. One grating was presented at 0° orientation and the other was presented at 90° orientation. Orientation was counterbalanced between stimulus locations across trials. All other properties of the gratings were the same as described for the evoked task above. One grating was presented in the receptive field area of the neurons recorded on the array, as described above, and the other grating was presented in the mirror symmetric location in the opposite hemisphere. The animals’ task was to detect a speed change (acceleration or deceleration) of one of the two gratings and to make a saccade to the stimulus that changed within 800 ms of the change onset. Correct responses were reinforced with juice or water. The speed change was governed by a triangular ramp function (that is, a gradual increase followed by a return to baseline, or vice versa). We titrated the maximum magnitude of the speed change in each direction before the experiment using a staircasing procedure to set an overall correct detection rate for each type of speed change between 70% and 80% correct. On 40% of trials, neither grating changed speed, and the animal was rewarded for maintaining fixation for the full stimulus duration, 1.2 s. The speed change, if any, could begin between 250 ms and 700 ms after grating onset, uniformly distributed. If a cue and speed change both occurred, the cue validly indicated the correct orientation of the target with 80% probability. The attention task design is shown in Supplementary Figure 3

While the animals performed the task, we recorded EEG data and spiking data as described above. Initial preprocessing (for example, EEG artifact rejection, spike identification and sorting) also proceeded as described above. We excluded from this analysis recording sessions with less than 72 correct responses to targets contralateral to the array (15 sessions in Monkey B and 4 sessions in Monkey R), because fewer responses produced highly variable estimates of $r_{ij}$. 

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We also excluded sessions for which an animal was both slower and less accurate for targets preceded by valid cues (zero sessions in Monkey B and 4 sessions in Monkey R), which indicated poor attention performance. This resulted in a total of eight recording sessions in the analysis for Monkey B, and a total of 34 recording sessions in the analysis for Monkey R. We also excluded trials with targets that occurred ipsilateral to the array, uncued trials, and trials with targets onsetting less than 500 ms after the grating onset or for which the animal prematurely left the fixation window, so that we always had a 500 ms interval of constant stimulation over which to compute alpha-band amplitude and \( r_{ac} \) (\( n = 91.25 \pm 7.18 \) trials per data set in the analysis for Monkey B; \( n = 179.56 \pm 8.30 \) trials per data set for Monkey R). We calculated alpha amplitude at the parieto-occipital EEG electrode over the array by multiplying the 500 ms of EEG data following grating onset by a Hanning window, taking the Fourier transform of the windowed data, and averaging the amplitude values over 8–12 Hz. We log transformed the alpha amplitudes to approximate a normal distribution. Because we were particularly interested in the effect of within-session variation in alpha amplitude, we calculated normalized amplitude by dividing each data set’s amplitude values by the mean amplitude for that session. We likewise normalized response times (RTs) and \( r_{ac} \) by dividing by the average for the session. We next binned each session’s trials into six quantiles based on alpha amplitude, and calculated mean RT, mean alpha amplitude and \( r_{ac} \) in each quantile. Values exceeding 3 s.d. from the mean were excluded as outliers (two data points excluded). We calculated \( r_{ac} \) by counting each unit’s spikes in the 500 ms interval following grating onset, standardizing each unit’s spike counts over trials separately for each grating orientation with a z-transform, and then calculating Pearson product-moment correlation over trials for each pair of units. We next used a regression analysis to quantify the relationship between alpha amplitude and RT, and the degree to which any such effect was mediated by \( r_{ac} \). Although the regression of RT on alpha amplitude could have in principle been performed using single trials, we used the means of the amplitude bins for this analysis so that the result could be meaningfully compared to a model that included \( r_{ac} \) which is only defined for sets of multiple trials. In line with our driving hypothesis, we first performed a second-order polynomial regression of RT on alpha amplitude in the ordinary least-squares sense using the following equation

\[
RT = \beta_{0}^a + \beta_{1}^a x + \beta_{2}^a x^2
\]

where \( x \) represents mean normalized alpha amplitude. Next, we added a linear term for \( r_{ac} \) again in line with our driving hypothesis

\[
RT = \beta_{0}^a + \beta_{1}^a x + \beta_{2}^a x^2 + \beta_{3}^a r_{ac} + \beta_{0}
\]

where \( y \) represents \( r_{ac} \). Since we were testing the specific model where RT follows a U-shaped relationship with alpha power and a direct linear relationship with \( r_{ac} \), we constrained \( \beta_{2}^a \) and \( \beta_{3}^a \) to each be non-negative.

We calculated the coefficient of determination \( R^2 \) for each model, and statistically tested the improvement in the proportion of variance-accounted-for by inclusion of the \( r_{ac} \) term against the null hypothesis that \( r_{ac} \) adds no information by using a bootstrap resampling procedure. For the bootstrap, we created 1,000 surrogate samplings of \( r_{ac} \) values by drawing at random an equal number with replacement from the original sample. For each bootstrap sample we repeated the bivariate regression described above and calculated \( R^2 \) to derive a distribution of \( R^2 \) values reflecting the null hypothesis. The proportion of the bootstrapped \( R^2 \) values exceeding the observed \( R^2 \) value is the \( P \) value for the statistical test.

Most critically for our predictions, we tested whether the inclusion of a linear term for \( r_{ac} \) led to a significant decrease in the magnitude of the quadratic term for alpha amplitude (that is, \( \beta_{2}^a \)). If so, then this would indicate that any specific U-shaped relationship between RT and alpha amplitude was mediated by the linear relationship between RT and \( r_{ac} \). The statistical significance for this test was assessed using a bootstrap resampling procedure as above. One advantage of a bootstrap procedure for this statistical test is that we compared the improvement in the model by including an additional term (that is, \( r_{ac} \)) for the observed data to the same improvement by including an additional term for the resampled data, which reflect the null hypothesis. Thus, significant differences between the observed and resampled data could not be simply due to having a greater number of model terms. We also performed this analysis for the other five frequency bands of interest besides the alpha band to test for frequency specificity.

A Supplementary Methods Checklist is available.

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