Chapter 3

Versatility of neuronal network function is maximized in the critical state

*In preparation*

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Abstract

Understanding why identical stimuli give differing neuronal responses and percepts is a central challenge in research on attention and consciousness. Synchronous activity in the form of ongoing oscillations on multiple scales of neuronal organization is thought to reflect functional states that bias the processing of incoming signals through its phase or amplitude. It is not known, however, whether these momentary phase or amplitude states depend on the long-term global dynamics and criticality of the networks generating the oscillations. Here, we use the framework of critical brain dynamics to investigate whether critical-state dynamics is important for pre-stimulus activity or stimulus strength to regulate post-stimulus responses.

We used a neuronal network model that exhibits ongoing oscillations with a level of criticality determined by the Excitation/Inhibition connectivity balance of the network. Networks were probed with different stimulus intensities and post-stimulus phase locking was analyzed in relation to pre-stimulus oscillation phase and amplitude as well as the strength of stimulation.

Our results show that three fundamental characteristics of neuronal network function have optimal versatility in the critical state: only networks with critical oscillations exhibit pre-stimulus amplitude and phase regulation of post-stimulus phase locking, and they show the largest dynamic range. Importantly, while the quantitative hallmarks of criticality require long-time monitoring of spatial and temporal dynamics that are statistically stable, the present results show that the concept of critical brain dynamics is compatible with time-varying functions—an important notion in contemporary theories of neuronal oscillations and their role in neuronal communication and attention.

Introduction

Understanding how neurons coordinate to produce emergent dynamics and behaviors is one of the crucial steps in answering how the brain gives rise to consciousness. To investigate this, groups have investigated the neural correlates of consciousness (Crick and Koch 1990; Engel and Singer 2001; Tononi and Koch 2008; Dehaene and Changeux 2011; van Gaal
and Lamme 2012). In addition to the neural correlates of consciousness there has been growing interest in understanding the neural pre-requisites of consciousness (Aru et al. 2012). Here, we aim to show how aspects of neuronal dynamics that have been proposed as pre-requisites and correlates of consciousness can be understood through the mechanism of critical brain dynamics.

The threshold-stimulus detection paradigm (Engen 1988; Linkenkaer-Hansen et al. 2004; Li et al. 2014; Wyart and Tallon-Baudry 2009) is widely used to investigate the neural correlates of consciousness. In these tasks, subjects are given a stimulus that is set at the edge of perception, meaning that on 50% of trials subjects perceive it, and on 50% of trials they do not. This allows you to compare neuronal activity between perceived and not perceived trials. Correlates of conscious perception in primary sensory regions have been associated with larger event-related potentials (e.g., the P100 component (Pins and Ffytche 2003)), larger event-related desynchronization (e.g., 8–24 Hz suppression (Vidal et al. 2015)), and stronger phase-locking to the stimulus (e.g., 1–30 Hz (S. Palva et al. 2005)). However, what causes some trials to evoke stronger ERPs, event-related desynchronization, or phase-locking than other trials—with dramatic consequences for perception—remains unclear.

To understand prerequisites of consciousness, researchers have analyzed how pre-stimulus activity affects the likelihood of a stimulus being detected or not. Both the momentary state of the network as defined by amplitude (Linkenkaer-Hansen et al. 2004; Händel et al. 2011; Ergenoglu et al. 2004; Hanslmayr et al. 2005) or phase (VanRullen et al. 2011) have been shown to bias stimulus processing. This state can be modulated by attentional mechanisms, and has led to the idea that alpha oscillations in the brain can serve to inhibit unwanted stimuli from reaching consciousness (Jensen and Mazaheri 2010; Jensen et. al 2012; Capilla et al. 2014).

Intriguingly, scale-free dynamics in oscillations and perception in threshold stimulus detection tasks are related (Palva et al. 2013), suggesting that critical-state dynamics matter for stimulus processing. Different computational models have associated critical dynamics in the form of neuronal avalanches to the largest dynamic range of evoked responses (Kinouchi and Copelli 2006; Shew et al. 2009). However, it is not known whether a similar result holds true for critical oscillations and oscillatory responses such as phase-locking.

In this study, we show that critical-state dynamics in a network is associated with versatile functions, allowing it to flow between low- and high-responsivity states on sub-second time scales. A critical network also shows the highest range of being able to respond differently to different stimuli strengths. Together, our results suggest that understanding how close neuronal networks are to criticality is essential for understanding their function.

Results

Unstimulated network produces multi-level criticality

To test the effect of critical oscillations on neuronal network functionality, we used a previously developed model of spontaneous neuronal activity (CROS) (Poil et al. 2012) with parameters optimized using an evolutionary algorithm (see Methods). This neuronal network model consists of 75% excitatory and 25% inhibitory integrate-and-fire neurons arranged in a 50x50 grid (Fig. 1A). The 2 parameters that need to be set when creating a network are the percentage of neurons within a local range (width 7 neurons) that each excitatory and each inhibitory neuron connects to (Fig. 1A). A power spectrum of the network activity signal showed a clear peak in the 8–16 Hz range (Fig. 1B) indicating oscillations. Looking across all of the networks, we see an increase in peak 8–16 Hz power with increasing Excitation/Inhibition (E/I) connectivity balance (Fig. 1C). Overall, this suggests that the amplitude of network activity oscillations is strongly linked to the E/I connectivity balance.

To characterize scale-free activity dynamics in the spatial domain, we analyzed neuronal avalanches and observed large variation in their distributions (Fig. 1D), with
Figure 1: CROS model displays multi-level criticality for balanced Excitation/Inhibition

A) Network consists of excitatory and inhibitory neurons arranged in a grid. Connectivity is set separately for excitatory and inhibitory neurons, and is defined as the percentage of neurons within its local range that each neuron connects to.
B) Network activity shows oscillations between 8–16 Hz. Shown for three example networks with low E/I connectivity balance (blue), medium E/I connectivity balance (green), and high E/I connectivity balance (red).
C) Oscillation power is dependent on E/I connectivity balance.
D) Spiking activity can display sub-critical (blue), critical (green) or super-critical (red) neuronal avalanches. Shown for same networks as (B).
E) Criticality of neuronal avalanches is dependent on E/I connectivity balance.
F) Oscillations can exhibit LRTC in their amplitude modulation. Shown for same networks as (B).
G) LRTC is dependent on E/I connectivity balance. (black line) indicates critical neuronal avalanches.
H) LRTCs peak with critical neuronal avalanches.
critical neuronal avalanches ($\kappa \approx 1$) occurring for a balanced E/I connectivity (Fig. 1E). Applying DFA to the network activity signal showed that there was non-trivial scaling behavior in 8–16 Hz amplitude fluctuations from 2–50 seconds (Fig. 1F). Comparing the scaling exponent across different networks (Fig. 1G) shows a clear peak in the scaling exponent. These results suggest that an E/I balance is necessary for long-range temporal correlations in oscillation amplitude to occur.

Comparing the two measures of criticality (avalanches and LRTC of oscillations), we find that the strength of LRTC peaks at critical neuronal avalanches ($\kappa \approx 1$) (Fig. 1H). This suggests that multi-level criticality, with spatial and temporal power-law scaling behavior in network activity, arises through a common mechanism of balanced E/I connectivity.

**Pre-stimulation amplitude influence on phase-locking requires LRTC**

We have seen that altering the E/I connectivity balance of a network changes its ongoing activity, but how does it affect a network’s response to a stimulus? To investigate this we attached a stimulus to 5 randomly chosen excitatory neurons. The ongoing oscillations showed a phase-locking response in the post-stimulus period (~65–250 ms) to the stimulus (Fig. 2A), which depended on the criticality of the network. This shows that the CROS model is capable of showing oscillatory responses to a stimulus, as has been seen in human subjects (S. Palva et al. 2005).

Considering the complex variation of activity in a critical network, we wondered whether critical networks function differently depending on their instantaneous state. For each run (with a stimulation strength, $n = 5$), we separated the trials into 10 separate bins based on pre-stimulus alpha power and then applied phase-locking analysis to each of these bins separately (Fig. 2B). This allowed us to analyze how pre-stimulus amplitude regulates the network’s response by calculating the correlation ($Reg_{amp}$) between the index of the bin (1–10) and the phase-locking response (Fig. 2C). A positive correlation means that high pre-stimulus amplitude leads to a strong phase-locking response at time point $t$, and a negative correlation means that low pre-stimulus amplitude leads to a strong phase-locking
Figure 2: Pre-stimulus amplitude regulation of response requires critical-state dynamics

A) Ongoing oscillations phase-lock to stimulus. Example shown for 5 neurons stimulated, for sub-critical (blue), critical (green), and super-critical networks (red).

B) Splitting trials from a critical network based on pre-stimulus power, shows different phase-locking response post-stimulus. Color indicates percentile of pre-stimulus amplitude bin.

C) Power of pre-stimulus oscillation can alter the phase-locking response of a network to the stimulus. Shown for example sub-critical (blue), critical (green), and super-critical (red) networks.

D) Networks tend to show no or a negative pre-stimulus regulation

E) The strength of pre-stimulus regulation is dependent on E/I connectivity balance. (black line) indicates critical neuronal avalanches.

F) Pre-stimulus regulation shows a significant correlation with LRTC in the latency range: 87–221 ms (Spearman correlation, red = p < 10^{-5}, Bonferroni corrected,)
response. Correlations values around zero mean that alpha power does not regulate the phase-locking response of the network. We found that sub-critical or super-critical networks tended to show no pre-stimulus amplitude regulation ($\text{Reg}_{\text{amp}} \approx 0$), whereas critical networks showed clear negative regulation (Fig. 2D). Looking across the connectivity parameter space, we found that for a balanced E/I connectivity there was strong negative pre-stimulus amplitude regulation (Fig. 2E). Correlating the strength of LRTC of the unstimulated network with the pre-stimulus regulation, we observed a significant negative relationship in a similar post-stimulus period (87–221 ms; Fig. 2F) to the overall post-stimulus phase-locking. This means that the stronger the LRTC, the more the pre-stimulus amplitude regulates the post-stimulus activity. With LRTC close to that of a randomly fluctuating oscillation, there is no pre-stimulus regulation of response. Overall, this means that the functional dependence on ongoing activity only holds for networks close to or at the critical state.

*Pre-stimulation phase influence on phase-locking requires LRTC*

Having identified a strong interaction between LRTC of oscillations and pre-stimulus amplitude on stimulus-evoked phase locking, we asked whether a similar effect is present for the phase of the pre-stimulus oscillation. To do this, we split the trials into 32 equally spaced overlapping bins (width $\pi/8$ radians) with equal number of trials in each bin. We then calculated the phase-locking response for all time points for the trials in each bin separately (Fig. 3A). If all bins show a similar phase-locking response at a time-point then there is no pre-stimulus phase regulation of response, whereas if there is a non-uniform distribution of phase-locking responses (e.g., bins close to $\pi$ show large PLF response, bins close to 0 show small PLF response), then there is pre-stimulus phase regulation of response (Fig. 3A). To quantify this, we calculated the uniformity of all the bins’ phase-locking response at time $t$, based on the pre-stimulus phase. For the sub- (blue) and super-critical (red) networks there is a similar post-stimulus phase locking for all pre-stimulus phase bins leading to no pre-stimulus phase regulation of response. For the critical (green) network there are large differences in post-stimulus phase-locking for different pre-stimulus
Figure 3: Pre-stimulus phase regulation of response requires critical-state dynamics

A) Phase of pre-stimulus oscillations can alter the phase-locking response of a network to a stimulus. Trials are split into 32 evenly spaced bins based on pre-stimulus phase (-5 ms) with an equal number of trials in each bin. Phase locking at all time-points can then be calculated for each bin. Pre-stimulus phase regulation of response is calculated based on the post-stimulus PLF distribution of the bins. Shown for example sub-critical (blue), critical (green) and super-critical (red) networks.

B) Pre-stimulus phase regulation is dependent on E/I connectivity balance. (black line) indicates critical neuronal avalanches.

C) Pre-stimulus phase regulation shows significant correlation with LRTC in the latency range: 120–171 ms (Spearman correlation, red $p < 10^{-5}$ Bonferroni corrected).
phase bins, meaning that the pre-stimulus phase regulates the post-stimulus phase-locking response.

Looking across the connectivity parameter space (Fig. 3B), we find that pre-stimulus phase dependence of post-stimulus phase locking requires balanced E/I connectivity almost identical to the balance required for critical oscillations to emerge. By correlating the strength of phase dependence at different latencies with the strength of LRTC of the unstimulated network, we find that there is a significant influence of a network’s LRTC on how the pre-stimulus phase regulates the post-stimulus phase locking in the 120–171 ms latency range (Fig. 3C). Therefore, the neuronal network function of pre-stimulus phase regulating post-stimulus response requires a critical network.

*Dynamic range of phase-locking response is strongest for critical networks.*

It is now clear that pre-stimulus oscillations can regulate post-stimulus activity, but what about differences in the stimulus itself? To test this, we investigated how changing the stimulus strength, $n$, altered the response of a network. In Figure 4A, we can see that a critical network increases the level of post-stimulus phase-locking as stimulus strength is increased. Post-stimulus response also depended on the criticality of the network (Fig. 4B), with stronger phase-locking happening in more sub-critical networks, and with more neurons stimulated. We calculated the dynamic range as the orders of magnitude of neurons stimulated over which a differential phase-locking response was produced (see Methods). The dynamic range gives an indication of how well a network can discriminate stimuli of different strengths. Across different networks, there was a significant correlation between post-stimulus dynamic range and the strength of LRTC of the unstimulated network (Fig. 4C,D). This shows that critical networks discriminate the widest range of stimuli through their post-stimulus phase locking response.
Figure 4: Dynamic range is maximized when network exhibits critical-state dynamics

A) Post-stimulus phase-locking response to a stimulus is dependent on the strength of the stimulus. Shown for different stimulation strengths of a critical network.
B) Dynamic range can be calculated by comparing the strength of phase-locking response to the number of neurons stimulated. Shown for example sub-critical (blue), critical (green) and super-critical (red) networks.
C) Dynamic range is dependent on E/I connectivity balance. (black line) indicates critical neuronal avalanches.
D) Dynamic range of phase-locking response is correlated with LRTC in the latency range: 65–542 ms (spearman correlation, red = $p < 10^{-5}$, Bonferroni corrected)
In this study, we showed for the first time a link between critical-state dynamics of oscillations and the ability of a network to respond differently depending on its ongoing activity. This functionality has been investigated in many human studies (Hanslmayr et al. 2005; Ergenoglu et al. 2004; Linkenkaer-Hansen et al. 2004), where a low oscillation power in relevant sensory areas in the pre-stimulus period has been seen as a pre-requisite of consciousness. In non-relevant areas, it has been suggested that a high oscillation power serves to functionally inhibit that region (Smith et al. 2012). Pre-stimulus phase has also been shown to bias perception (VanRullen et al. 2011) and we found that this function is also likely to be modulated by the criticality of the network. In addition, we show that oscillatory spiking neuronal networks have an optimal dynamic range when they are critical, which is in agreement with previous work on non-oscillatory networks (Kinouchi and Copelli 2006; Shew et al. 2009), and which suggests that the largest dynamic range is a generic property of a critical system.

Pre-stimulus activity regulation of post-stimulus response has been found in different stimulus detection experiments, and has shown to be effective in terms of enhancing detection of stimuli that are attended to (Capilla et al. 2014) or are expected (Mayer et al. 2015), and reducing detection of irrelevant stimuli (Händel et al. 2011). However, is this network ability always optimal for performing a task? In tasks where continuous detection ability is required then fluctuations in pre-stimulus activity will actually decrease task performance in those periods of amplitude or phase that produce smaller post-stimulus response. This has been shown in reduced phase-locking to a stimulus when people experience periods of mind-wandering when performing a task (Baird et al. 2014). In these cases a more-sub-critical network would actually perform better in terms of constant attention (Tomen et al. 2014) and a super-critical network would perform better in terms of constant functional inhibition. Therefore, we propose that mind-wandering in tasks could be strongly related to the criticality of the network.

There is much debate as to the cause of amplitude fluctuations during threshold stimulus detection tasks, with top-down mechanisms such as attention and expectation
being suggested (Melloni et al. 2011; Jensen et al. 2012). Here, we show that a network with balanced excitatory and inhibitory connectivity produces amplitude fluctuations that have a functional role, meaning that the network goes through periods of high and low sensitivity to external stimuli. In the unbalanced state, the network loses this function and either reliably reacts (in the sub-critical case), or ignores the stimulus (super-critical case). Thus, top-down mechanisms are not required for fluctuations in alpha power; however, it is plausible that attentional mechanisms can actively regulate the level of fluctuations to shift the network’s operating point relative to the critical state to react in the required manner (Wyart and Tallon-Baudry 2009).

The relationship between criticality and pre-stimulus regulation of post-stimulus response fits well with existing literature showing that LRTC correlate with the scaling relationship in conscious perception (Palva et al. 2013). We propose that this correlation is rooted in a mechanism where fluctuations in the pre-stimulus amplitude regulate evoked responses and their perceptual consequences (Palva et al. 2005).

Dynamic range of different aspects of network activity such as average activity (Kinouchi and Copelli 2006), and number of evoked nLFP (Shew et al. 2009) has been shown to be maximized in the critical state. It still remains unclear whether this is a generic property of neural evoked responses. Here, we showed that the maximum dynamic range can also apply to oscillatory responses in the form of phase-locking to the stimulus. This raises the possibility that other aspects of evoked responses will be affected by the level of criticality such as different time components of the event-related potential.

A sensory network is embedded into large-scale networks, and we propose that the criticality of a local network will alter how it is functionally connected into the large-scale network. It has been shown that the pre-stimulus connectivity state of a network also alters the chance of a stimulus being detected (Weisz et al. 2014; Leske et al. 2015), and it has yet to be seen how criticality affects this.
Overall our study highlights the importance of critical-state dynamics (Chialvo 2010) for understanding neuronal network function, and shows that it is possible to integrate oscillatory mechanisms into this framework.

Methods

Critical Oscillations (CROS) model

We modeled networks of 75% excitatory and 25% inhibitory integrate-and-fire neurons arranged in a 50x50 open grid. Networks differ in their two connectivity parameters, which are the percentage of other neurons within a local range (width = 7 neurons) that each excitatory and each inhibitory neuron connects to (Fig. 1A). Connectivity parameters were set between 25–100% at 5% intervals. Border neurons had fewer connections, because these neurons had a lower number of neurons in their local range. Connectivity was probabilistic and decreases with distance with an exponential decay. More specifically, the probability, P, of a connection at a distance r was given by:

\[ P(r) = Ce^{-r} \]  

where C is a constant determining the connection probability.

Neuron model

Neurons were modeled using a synaptic model integrating received spikes, and a probabilistic spiking model. Each time step (dt) of 1 ms starts with each neuron, i, updating \( I_i \) with received input from any of the set of connected neurons (J), together with an exponential synaptic decay, which can either be excitatory or inhibitory.

\[ \tau_I \frac{dI_i}{dt} = I_0 - \left( I_i + \sum_j W_{ij} S_j \right) \]  

S is a binary spiking vector of the neurons spiking in previous time step, and weights are fixed depending on the type of the pre- and post-synaptic neuron.
The probability of spiking, \( P_S \), is then updated with this input, together with an exponential decay:

\[
\tau_p \frac{dP_{Si}}{dt} = P_0 - (P_{Si} + I_i) \quad (3)
\]

We determine whether the neuron spikes with the probability \( P_S \), and update the spiking vector for the next time step. If a neuron spikes, the probability is reset to the reset value \( P_r \) and the binary spiking vector \( S \) is updated. In the next time step, all neurons that it connects to will have their input updated according to Equation 2.

**Model Parameters**

All of the parameters for the model were the same as the original paper (Poil et al. 2012) apart from the synaptic weights. Neuron model: \( (\tau_I = 9 \text{ ms}, I_0 = 0) \). Synaptic model: Excitatory neurons \( (T_P = 6 \text{ ms}, P_0 = 0.000001 [1/\text{ms}], P_r = -2 [1/\text{ms}]) \) and inhibitory neurons \( (T_P = 12 \text{ ms}, P_0 = 0, P_r = -20 [1/\text{ms}]) \).

To improve the range and stability of the long-range temporal correlations from the original model an evolutionary algorithm (Smit and Eiben 2011) was applied to the synaptic weights. The parameters that could vary were the 2 connectivity parameters (taking values between 0–100%) and the natural logarithm of the magnitude of the 4 synaptic weights (taking values between -5 and 1). For each run, a fitness values was calculated based on the avalanches size and duration distributions and the LRTC.

\[
\text{fitness} = \frac{1}{|1-\text{DFA}| + |1-\kappa_{\text{size}}| + |1-\kappa_{\text{duration}}|} \quad (4)
\]

The optimum weights \( (W_{ij}) \) found by the algorithm were \( \{W_{EE} = 0.0085, W_{EI} = 0.0085, W_{IE} = -0.569, W_{II} = -2\} \).

**Network activity analysis**

A network signal was created by summing the total number of neurons spiking at each time-step with a Gaussian noise signal of the same length with mean = 0 and \( \sigma = 3 \). The
level of white noise was set to allow all networks to achieve a time-varying phase which is not the case without adding noise when there are silent periods in the network.

**Connectivity Parameter spaces**

To display connectivity parameter spaces, values are linearly interpolated at 0.5% connectivity intervals.

**Oscillation power**

To estimate the peak frequency, we applied a power spectrum estimate (Welch method) using a hamming window with $2^{11}$ FFT points. Power in a band was calculated by summing the power values between the frequency bands.

**Detrended fluctuation analysis of long-range temporal correlations.**

Detrended fluctuation analysis (DFA) was used to analyze the scale-free decay of temporal (auto)correlations, also known as long-range temporal correlations (LRTC). The DFA was introduced as a method to quantify correlations in complex data with less strict assumptions about the stationarity of the signal than the classical autocorrelation function or power spectral density (Linkenkaer-Hansen et al. 2001; Hardstone et al. 2012). An additional advantage of DFA is the greater accuracy in the estimates of correlations, which facilitates a reliable analysis of LRTC up to time scales of at least 10% of the duration of the signal (Chen et al. 2002; Gao et al. 2006). DFA exponents in the interval of 0.5 to 1.0 indicate scale-free temporal correlations (autocorrelations), whereas an exponent of 0.5 characterizes an uncorrelated signal. The main steps from the broadband signal to the quantification of LRTC using DFA have been explained in detail previously (Linkenkaer-Hansen et al. 2001; Hardstone et al. 2012). In brief, the DFA measures the power-law scaling of the root-mean-square fluctuation of the integrated and linearly detrended signals, $F(t)$, as a function of time window size, $t$ (with an overlap of 50% between windows). The DFA exponent is the slope of the fluctuation function $F(t)$, and can be interpreted as the strength of the autocorrelations in signals.
**Neuronal Avalanches**

Spatial avalanches of activity were analyzed by summing the total number of neurons spiking at each time-step, and defining avalanches as periods of activity that were above threshold (set at 0.5 * median number of neurons spiking at each time-step). The size of an avalanche is the total number of neurons that spike between two periods of sub-threshold activity. To assess the scale-free nature of the avalanche size distribution (Beggs and Plenz 2003) we quantified the similarity between the distribution of our data and a power-law by calculating the average difference of the cumulative distribution of a power-law function with an exponent of -1.5 and that of our experimental data at 10 equally spaced points on a logarithmic axis and adding one. This gives the κ index (Shew et al. 2009). A subcritical distribution is characterized by κ < 1, and supercritical distribution by κ > 1, whereas κ = 1 indicates a critical network.

**Excitation/Inhibition connectivity balance**

The variable parameters of a CROS network are the excitatory and inhibitory connectivity. The Excitation/Inhibition connectivity balance is defined as the combination of excitatory and inhibitory connectivity parameters.

**Unstimulated networks**

To assess the dynamics of unstimulated networks we allowed connectivity to take values between 25–100% at 5% intervals. For all 256 possible parameter combinations of excitatory and inhibitory connectivity, we created 20 different networks and ran each network for $10^6$ time-steps (1000 seconds).

**Stimulation networks**

To test network response to a stimulus we took one sample network for each combination of excitatory and inhibitory connectivity parameters, and for each run of the network attached a stimulus to 5 randomly chosen excitatory neurons. To pick the network to stimulate for each combination of excitatory and inhibitory connectivity—from the 20
unstimulated networks run for each combination of connectivity—the networks were ranked and scored based on distance from median rank on: (Mean network activity, Standard deviation network activity, $\kappa_{\text{size}}$, $\kappa_{\text{duration}}$, DFA exponent (2–10 seconds), DFA exponent (2–50 seconds)). These ranks were summed and the picked network was the one with the lowest summed rank meaning it was the closest to being the median network for those parameters.

During the stimulation run of $2 \times 10^6$ time steps of 1 ms these neurons simultaneously received a stimulus every 750–1250 ms. The stimulus had the same weight as the existing excitatory-excitatory connections in the network.

**Phase-locking factor**

Stimulus response was calculated in the network in terms of the phase-locking factor (Makeig et al. 2002; Palva et al. 2005; Hirvonen and Palva 2015). Data were filtered using a fir filter (order 0.25) between 8–16 Hz and the phase obtained by taking the angle of the Hilbert transform. Phase-locking factor (PLF) measures the uniformity of phases across trials at a time-point post-stimulus and was calculated using the circstats toolbox (Berens 2009).

**Pre-stimulus amplitude regulation of PLF**

To calculate the pre-stimulus amplitude regulation of PLF, trials were separated into 10 percentile bins, based on the pre-stimulus amplitude in the 8–16 Hz frequency range in the time-range -150 to -50 ms, and PLF calculated separately for each bin. The regulation measure r was calculated as the Pearson correlation between the index of the bin and the PLF for that bin at time point t.

**Pre-stimulus phase regulation of PLF**

To calculate the pre-stimulus phase regulation of PLF, trials were split into 32 overlapping bins of width $\pi/8$ based on the pre-stimulus phase in the 8–16 Hz frequency range at time -5 ms. To get an equal number of trials in each bin, x randomly selected trials from each bin
were picked where x was the smallest number of trials in a bin. Post-stimulus PLF was calculated separately for each bin. To check the uniformity of the distribution of post-stimulus PLF for the bins, we calculated the mean resultant vector length of the normalized distribution giving a value between 0 (uniform circular distribution) and 1 (all PLF in one bin). We also performed this procedure for the unstimulated network, and subtracted this PLF from the PLF of the stimulated network to get a measure of the stimulus-induced pre-stimulus phase regulation.

Dynamic Range

To calculate the dynamic range of a network, we stimulated n neurons where n ∈ {1, 2, 3, 5, 9, 15, 25, 43, 74, 126, 216, 369, 632, 1081, 1800} and fitted a sigmoidal curve to the PLF response for n. The dynamic range was calculated as the orders of magnitude of neurons stimulated between the 10% and 90% PLF response (Kinouchi and Copelli 2006).

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