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Spike counts in the visual cortex consistently encode both stimuli and behavioral choices in a change-detection task

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In visual discrimination tasks, the subject collects information about sensory stimuli and makes behavioral decisions accordingly. In this study, we are searching for coding strategies in visual cortices of the macaque (*macaca mulatta*) that relate to both stimuli and behavior. Multi-units within a single cortical column are recorded in V1 and V4 areas simultaneously while the subject is performing a change detection task with matching and non-matching stimuli. We assess systematic differences in distribution of spike counts for matching vs. non-matching stimuli (detection probability) and for correct vs. incorrect behavioral performance (choice probability, [1]) on the single cell and on the population level. In addition, we estimate pair-wise correlations of spike counts. The spiking signal is weakly but significantly predictive on the type of stimulus (matching vs. non-matching stimuli with correct behavioral responses) as well as on different behavioral choices with correct and incorrect behavioral performance (correct vs. incorrect behavioral responses on non-matching stimuli). In both areas, the effect is limited to the superficial layers of the cortical column. Detection and choice probability are consistent, the behavioral choice "match" being characterized by higher spike counts in both cases. In V1, but not in V4, the signal corresponding to the choice"match" is even statistically invariant with changes in both the type of the stimulus and the behavioral performance. In incorrect trials, neural activity in V1 is in addition characterized by a systematic bias in spike counts already at the beginning of the trial. The bias is consistent with the future behavioral choice and is only present in the deep cortical layers. Comparing the distribution of correlation coefficients across pairs of neurons with matching and non-matching stimuli, distribution of coefficients in V4 is less variable with matching stimuli, in particular for short (0-0.5 mm) and middle-range (0.5-1 mm) inter-neuron distances. This effect could be interpreted as a fast adaptation of neural responses to two consecutive presentations of the same stimuli [2]. A change in long-range (>1 mm) correlations in V4 is observed when comparing trials with correct and incorrect behavioral performance, correlations in incorrect trials showing higher variability. In V1, we did not observe any systematic changes in spike-count correlations with different stimuli. However, correlations are significantly more variable in trials with incorrect compared to correct behavioral performance. This effect is once again limited to deep cortical layers. Higher variability of correlations in V1 might be a signature of spontaneously generated network state that is more likely leading to incorrect behavioral performance. Finally, we test the interactions between choice probabilities and spike-count correlations. Choice probabilities and correlations do not interact in V1, but weakly interact in the V4 area, where cells with similar choice probabilities tend to be more strongly correlated. In summary, we observe various differences in the first and second order statistics of spike counts in both V1 and V4 areas. The first order statistics is related to coding of both stimuli and behavioral choices while correlations would rather modulate the efficacy of encoded signals.

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Local topology of connectome stabilizes critical points in mean field model

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The interplay between structural connectivity (SC) and neural dynamics is still not yet fully understood. Applying topological analysis, the connectome approach links this anatomical network to brain function. Here we adopt a computational approach to find topology features related to the stability on global neural dynamics. A previous study of a mean field model based on the human cortex network, shows at least 2 global neural states, with either a low or high firing rate pattern [1, 3]. These 2 possible states, or bistability, emerge in the model within a range of the global coupling parameter G, limited by critical values G_- and G_+[1, 3]. Also, at this bistable range, this model achieves the highest correlations with empirical resting state fMRI data. How the network connectivity pattern shapes the critical G values has not been yet investigated. Our aim is to identify local or global topology features related to the critical G values. We studied 4 different SC networks: a cortical parcellation of human brain [2], a human binary equivalent, a Random Network (RN) having the same degree distribution as human SC, and an equivalent Watts & Strogatz Small World (SW) network. For each of the analyzed networks, values in their critical G points have small or null variability. Then, we selectively prune the edges of the networks and calculate their critical G values to show the effect of structure pattern in maintaining the bistable dynamics. The edges were pruned selectively based on either the degree or the k core decomposition measure; interpreted as a local or global topology feature, respectively. Also, the pruning procedure is applied to the edges on one of 3 specific ways: i) high degree/k core nodes, ii) random cuts, and iii) low degree/no k core nodes. The highest shifts in critical G values are achieved when the edges of high degree or k core nodes are pruned. In contrast, when we prune those edges belong to low degree or no k core nodes, the shifts in the critical G points are irrelevant. We interpret this as that the model can use either local or global connectivity pattern in order to stabilize the critical G points. Furthermore, our study show that shifts in the critical G points are statistically equivalent when the degree distribution (but not k core structure) is shared, such as in the binary human SC compared to the RN. Therefore, in our simulation the degree distribution, interpreted as a local connectivity feature, determines the critical G points for bistability, capturing the essential structural pattern of the network. We also show that it is possible to obtain bistability in other types of networks, suggesting that structure dynamic relationships may obey a topological principle.

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How chaos in neural oscillators determine network behavior

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Chaotic dynamics of neural oscillations has been shown at the single neuron and network levels, both in experimental data and numerical simulations. Theoretical works suggest that chaotic dynamics enrich the behavior of neural systems, by providing multiple attractors in a system. However, the contribution of chaotic neural oscillators to relevant network behavior has not been systematically studied yet. We investigated the synchronization of neural networks composed of conductance-based neural models that display subthreshold oscillations with regular and burst firing [1]. In this model, oscillations are driven by a combination of persistent Sodium current, a hyperpolarization-activated current (Ih) and a calcium-activated potassium current, very common currents in the CNS. By small changes in conductance densities, the model can be turned into either chaotic or non-chaotic modes [2]. We study synchronization of heterogeneous networks where conductance densities are drawn from either chaotic or non-chaotic regions of the parameter space. Measuring mean phase synchronization in a small-world network with electrical synapses, we characterize the transition from unsynchronized to synchronized state as the connectivity strength is increased. First, we draw densities from fixed-size regions of the parameter space and find the transition to synchronized oscillations is always smooth for chaotic oscillators but not always smooth for the nonchaotic ones. However, non-smooth transitions were found to be associated to a change in firing pattern from tonic to bursting. Nevertheless, we noticed that chaotic oscillators display a wider distribution of firing frequencies than non-chaotic oscillators, thus making more heterogeneous networks. Next, we draw the conductance densities from the parameter space in a way that maintained the same distribution of firing frequencies (hence the heterogeneity of the network) for both chaotic and non-chaotic. In this case, synchronization curves are very similar, being second order phase transition for both cases. However, we cannot discard that non-chaotic oscillators become chaotic (or vice versa) when in a network, because of the extra parameter associated to the electrical synapse. Finally, when the chaos-inducing Ih current is removed, the transition to synchrony occurs at a lower value of connectivity strength but with a similar slope.
Our results suggest that the chaotic nature of the individual oscillators may be of minor importance to the synchronization behavior of the network. Ongoing work is being conducted to measure the chaotic nature of the whole network, and how it is related to the synchrony behavior.

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STEPS 3: integrating stochastic molecular and electrophysiological neuron models in parallel simulation

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Stochastic spatial molecular reaction-diffusion simulators, such as STEPS (STochastic Engine for Pathway Simulation) [1], often face great challenges when simulating large scale complex neuronal pathways, due to the massive computation required by the models. This issue becomes even more critical when combining with cellular electrophysiological simulation, one of the main focuses in computational neuroscience research. One example is our previous research on stochastic calcium dynamics in Purkinje cells [2], where a biophysical calcium burst model was simulated on approximate ¼ of a Purkinje cell dendritic tree morphology using the serial implementation of spatial Gillespie SSA and electric field (EField) solver in STEPS 2.0. Even with a state-of-the-art desktop computer, it still took months to finish the simulation, significantly slowing down research progress.
One possible, yet not trivial approach to speedup such simulation is parallelization. In CNS2016 we reported our early parallel implementation of an Operator-Splitting solution for reaction-diffusion systems, which achieved super-linear speedup in simulation of the buffer components of the above published model on full Purkinje cell morphology. While the performance of our parallel implementation was promising, the test model had no calcium presented in the system and only buffers were simulated. Since buffers were uniformly distributed in the geometry, the loading of each computing process was relatively balanced, resulting in a close to ideal scenario for parallel computation. The membrane potential computation, as well as voltage-dependent reactions in the published model, were omitted due to the lack of a parallel EField solver at the time. In a recent publication [3], we further extended the model by applying a dynamically updated calcium influx profile extracted from the published calcium burst simulation. Our result shown that in a realistic scenario with dynamic calcium influx, data recording, and without special load balancing, our parallel reaction-diffusion solution can still achieve more than 500 times of speedup with 1000 computing processes comparing to the conventional serial SSA solution.
STEPS 3 is the first public release out of the collaboration between the CNS Unit of OIST and the Blue Brain Project of EPFL. The ongoing collaboration aims to deliver a scalable parallel solution for future integrated stochastic molecular and electrophysiological neuron modelling. Combining the parallel TetOpSplit molecular solver developed by OIST and EPFL's parallel EField solver based upon the PETSc library, our new release addresses the limitations of above test cases, and allows full scale parallel simulation of the complete Purkinje cell calcium burst model. It also contains new changes that are essential to parallel STEPS