Transition to chaos in heterogeneous networks

Johnatan Aljadeff[1], Merav Stern[2], Tatyana O. Sharpee[1]

Abstract. There is accumulating evidence that biological neural networks posses optimal computational capacity when they are poised at or near a critical point where the network transitions to a chaotic regime. A majority of theoretical models that predict when a network becomes chaotic consider homogeneous (or nearly homogeneous) networks, but real neural networks are heterogeneous with respect to cell types and connectivity patterns. We derive a mathematical condition for networks with heterogeneous connectivity rules to become chaotic. We apply our results to networks where adult neurogenesis occurs and to clustered networks and show that our theoretical model may provide insight into their structure-function relationship.

Results. In this study we derived the condition for a network with multiple cell types to cross a critical point and become chaotic. To define the network, we first divide the N neurons into D types, each with N_d neurons ($\sum_{d=1}^{D} N_d = N$). The connection weights between every pair of neurons is randomly drawn from a distribution that depends on the type identity of both neurons. These distributions are summarized in a $D \times D$ non-negative *connectivity rule matrix* G, that does not need to be symmetric. Each element J_{ij} of the full connectivity matrix is drawn from a centered distribution with variance $G_{c_i d_j}/N$ where c_i and d_j are the type indices of neurons i and j, respectively. The network obeys the standard rate dynamics $\dot{x}_i = -x_i + \sum_{j=1}^{N} J_{ij} \tanh x_j$.

In the special case of a homogeneous network (consisting of a single cell type), the connection weights are all drawn from a distribution with variance G/N. Here, the eigenvalues of the matrix J are distributed inside a circle of radius $\sqrt{G}$ in the complex plane (Girko's circular law). Sompolinsky et al. [1] proved that this network becomes chaotic for $G > 1$. We show that, similarly to the homogeneous case, a heterogeneous network undergoes the same transition when eigenvalues of its connectivity matrix exceed one in absolute value. To fully characterize the transition of the network from a single fixed point to chaos, we derived a formula (in terms of the matrix G and the vector N_d) for r, the boundary of the spectrum of the heterogeneous connectivity matrix. r is the square root of the maximal eigenvalue of a deterministic $D \times D$ matrix M whose c, d element is $M_{cd} = \frac{1}{N} N_c G_{cd}$.

This allows us to analytically relate the criticality of the network to the parameters describing its heterogeneity. We study two examples of heterogeneous networks, and show how our formula can give insights into their structure-function relationship. Before a brief discussion of these examples, note that our analysis also enables us to determine the relative activations of different neuron types in the chaotic regime; and that our formula is not simply the mean variance across all connections.

Example 1, Adult neurogenesis. We apply these results in the context of networks where new hyperexcitable neurons are continuously integrated. We find the scaling relationship between the fraction of new cells α and their relative hyperexcitability γ. When $\alpha\gamma^4 \gtrsim 1$ the network becomes chaotic and therefore can be efficiently trained via a supervised learning algorithm [2]. The learnability is modulated primarily by the radius of the spectrum we derived. Our analysis suggests the role of the new neurons is context dependent.

Example 2, Clustered networks. We can define each cell type to be a cluster of neurons, with strong intra- and weak inter-cluster connectivity. When the number of clusters is large, even when

[1]Computational Neurobiology Laboratory, The Salk Institute for Biological Studies; Center for Theoretical Biological Physics and Dept. of Physics, Univ. of California, San Diego

[2]Dept. of Neuroscience, Columbia Univ.; Interdisciplinary Center for Neural Computation, Hebrew Univ.

the overall connection strength in the network is constant, small asymmetries in the cluster sizes can lead to a transition to the chaotic regime. In the limit of weak inter-cluster connectivity, small deviations in cluster sizes lead to a switching effect in the identity of the dominating cluster.

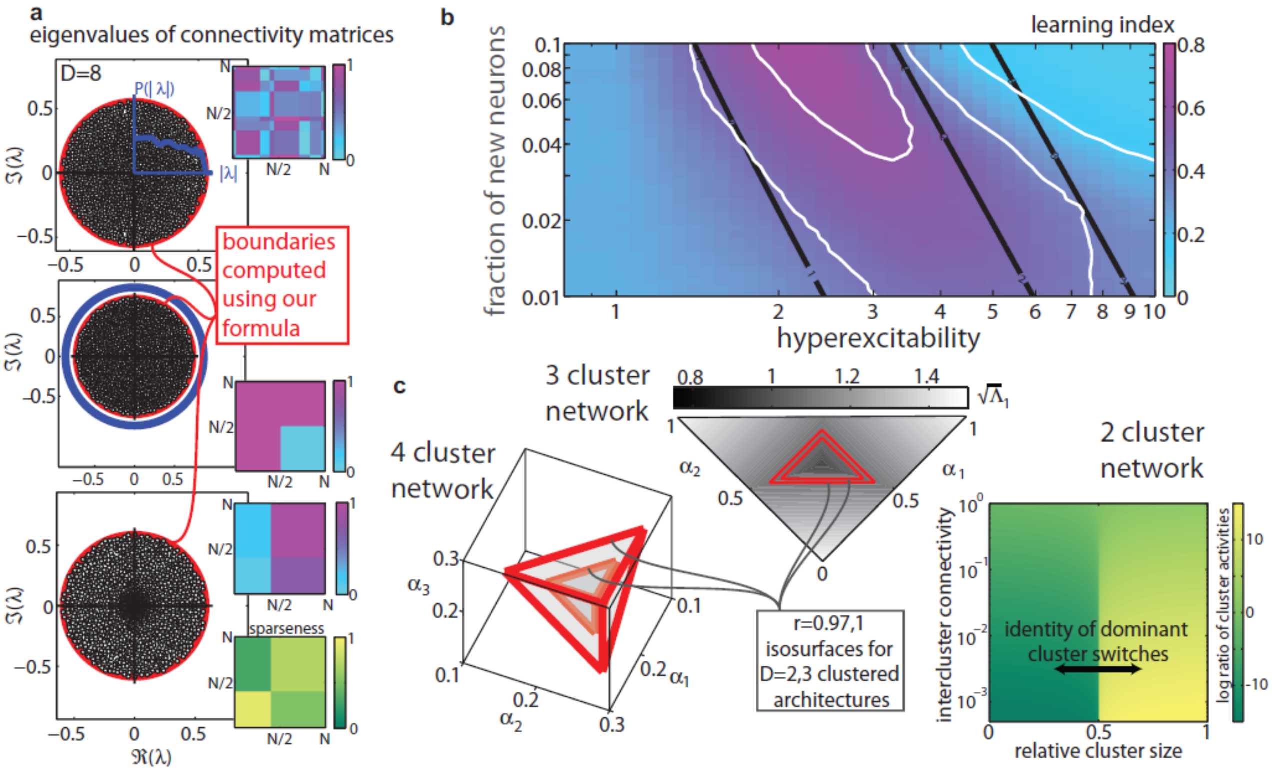

Figure 1: (a) Example eigenvalue spectra for three networks plotted in the complex plane. Connection strength between neurons belonging to sub-populations and the relative size of each sub-population is indicated in blue-purple. Sparseness (only for bottom panel) is in green-yellow. Top - the boundary of the spectrum of a network with $D = 8$ cell types (and 64 distinct connectivity rules) is in good agreement with our formula (red). The radial distribution (in blue) vanishes at the boundary we predicted. Middle - a network with $D = 2$ cell types chosen to highlight the difference between our formula and the naive guess given by the mean variance (blue). Bottom - our formula can be extended to include sparse networks or other non-gaussian connectivity distributions. (b) The learning index (the convolution of target and output signals) of the neurogenic network model averaged over four target frequencies as a function of r. Contour lines of the learning index (white) and the radius (black) approximately coincide. (c) In a clustered architecture with 3,4 clusters, isosurfaces of the radius of the spectrum as a function of the cluster size form tetrahedra (triangles). In the limit of large number of clusters the volume within the isosurface $r = 1$ vanishes, suggesting that small heterogeneity in cluster sizes may lead to a phase transition. For a two cluster network, in the limit of weak inter-cluster connectivity a switching effect is observed where small fluctuations in cluster size lead to dramatic changes in the relative activity of the clusters.

References

[1] H. Sompolinsky, A. Crisanti, and H. Sommers, Physical Review Letters **61**, 259 (1988).

[2] D. Sussillo and L. F. Abbott, Neuron **63**, 544 (2009).