Beyond balance: The role of network structure in population dynamics—Elan Liss Ohayon[1,3], Maxim Bazhenov[1], Terrence J. Sejnowski[1], Hon C. Kwan[2], W. McIntyre Burnham[3] ([1]Computational Neurobiology Laboratory, Salk Institute, La Jolla, CA, USA, [2]Department of Physiology, University of Toronto, Toronto, ON, Canada, [3]University of Toronto Epilepsy Research Program, Toronto, ON, Canada)

Balance of synaptic excitation and inhibition as well as intrinsic neuronal properties are often cited as the central factors in determining the persistence of brain activity and propagation. In this study, we use computational models to demonstrate that the structure of a network, such as the level of heterogeneity in connection patterns, can be a critical feature in determining network dynamics.

To illustrate the phenomenon we simulated networks with up to 5000 spiking neurons using a difference equation based model. The networks consisted of both inhibitory and excitatory populations with inter-layer and columnar intra-layer connectivity. We then show that the inclusion of heterogeneous connectivity patterns—in the form of network boundaries, diffuse cell removal or localized cell deletion—can quantitatively and qualitatively affect network threshold response, activity duration and propagation patterns. As an example, we began with an intact homogeneous network in which the response to a localized stimulus or distributed noise was shown to dissipate quickly. Cells were then randomly removed and connectivity density lowered (p deletionZ0.3–0.7). Following the deletions, the same stimulus level now resulted in persistent activity in the form of propagating waves. As density was lowered further (p deletionZ0.7–0.9) the wave propagation patterns were progressively reduced and eventually disappeared. That is, the persistent activity remained but the network became too sparse to support waves. Spatial activity in the sparse networks tended to remain localized and settle into isolated pockets of oscillatory patterns. The variation in activity as a function of changes in network structure was thus non-monotonic.

It is important to note that the inhibitory to excitatory ratio of the connections was kept constant as were cell intrinsic properties. The model thus shows that connectivity structure can determine population activity independently of changes to inhibitory and excitatory balance. The findings suggest that architectural alterations may be important for understanding threshold drops seen in post-traumatic epilepsy. The findings are also relevant to changes in EEG seen in neurodegenerative disorders. Most importantly perhaps, network structure is clearly a critical factor in considering how the normal brain maintains the persistent activity required for cognitive processing.