Heterosynaptic plasticity rules induce small-world topologies

James McAllister[1], John Wade[1], and Cian O'Donnell [1]

[1] *Intelligent Systems Research Centre, Magee Campus, Ulster University*

Abstract: Heterosynaptic plasticity is a form of 'off-target' synaptic plasticity where unstimulated synapses change strength. Although some theoretical work has explored its implications [2], its function for brain learning remains unknown. Here we propose that one purpose of heterosynaptic plasticity is to encourage small-world brain connectivity [4, 5]. We used numerical computer simulations to compare different plasticity rules in fully-connected abstract weighted graphs, and found that they yield distinct network architectures. Simple heterosynaptic plasticity-based learning – in contrast to Hebbian-style homosynaptic learning – not only reduces the over-saturation of synaptic weights, but cause the networks to rapidly converge to small-world topologies.

The simplest version of the model is constructed as follows: We initialise a fully connected and symmetric mathematical graph, consisting of N nodes (abstracted to represent single neurons, or groups of neurons) and weighted relations $w_{i,j}$ between the i^{th} and j^{th} nodes (interpreted as interneural synaptic weights). In this simplest model, the nodes have no self connections. This may be represented by a hollow symmetric weight matrix.

At every iterative step, a subset S of nodes is sampled (where nodes in S may be taken to be neurons/populations which are "active"). We explore different constructions of the subset S, from consisting of sets of constant members and constant size, to sets of varying size with probabilities dependent on the weights associated with each node. (The probability of a node i being in S in this final case is defined as $\frac{\sum_i w_{i,j}}{N-1}$. This preferentially selects nodes with higher accumulations of weights). With this setup of S, there are three cases: each weight is either (1) between two active nodes, (2) between one active and one inactive node, or (3) between two inactive nodes. Based on these cases, 2 plasticity rules are defined:

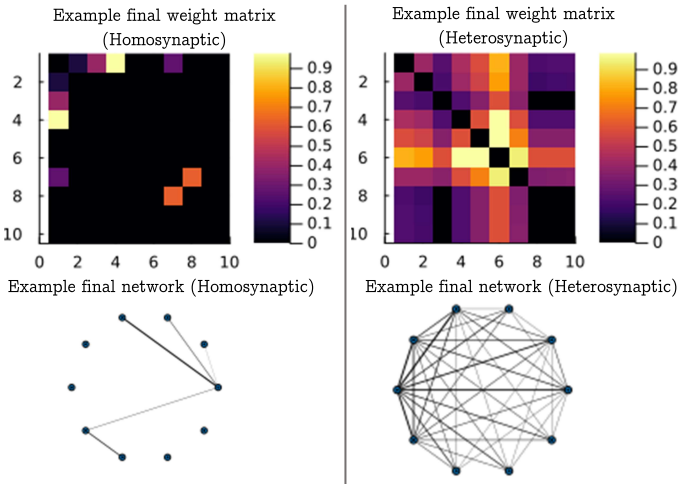

Figure 1: Final homosynaptic weight matrix and network (left), Final heterosynaptic weight matrix and network (right)

$$R_1 = \left\{ \begin{array}{ll} w_{i,j_{n+1}} = w_{i,j_n} + \eta_1(1 - w_{i,j_n}) & \text{if } i \text{ and } j \in S \\ w_{i,j_{n+1}} = \eta_2 w_{i,j_n}, & \text{otherwise} \end{array} \right\} \quad (1)$$

$$R_2 = \left\{ \begin{array}{ll} w_{i,j_{n+1}} = w_{i,j_n} + \gamma_1(1 - w_{i,j_n}) & \text{if } i \text{ and } j \in S \\ w_{i,j_{n+1}} = w_{i,j_n} + \gamma_2(1 - w_{i,j_n}) & \text{if } i \in S \text{ and } j \notin S \\ w_{i,j_{n+1}} = \gamma_3 w_{i,j_n}, & \text{otherwise} \end{array} \right\} \quad (2)$$

R_1 updates the weights in the network by a simple rule which strengthens weights between co-active nodes ("those that fire together, wire together"), and weakens all others where η_1 and η_2 are constants determining the strengthening and weakening of weights, respectively. R_2, the second, heterosynaptic rule is defined so as to strengthen weights between sets of co-active nodes, partially strengthen weights between partially active sets of nodes, and weaken others. where γ_1, γ_2 and γ_3 denote the constants of heterosynaptic strengthening (γ_1, γ_2) and weakening (γ_3). Without loss of generality, the formulation of these above learning rules ensures that the weights are bounded between the values of 0 and 1. When a weight becomes sufficiently small (i.e. $w_{i,j} < 0.1$), it is set to equal zero, denoting the total loss of a synaptic connection.

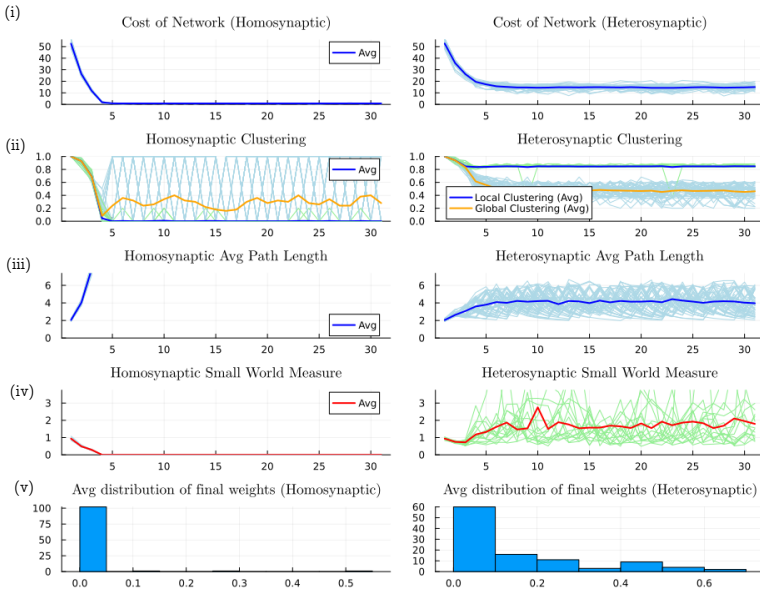

Figure 2: Small-world network measures across 100 trials; Homosynaptic plasticity rule (left), Heterosynaptic plasticity rule (right)

Across simulations of the above model, various graph theoretic measures are calculated such as local and global clustering coefficients (C), average shortest path length (L), and the overall weight "cost" of the network. Small-world characteristics are difficult to definitively measure, however, in this work we calculate a coefficient for small-world measure as $\sigma = \frac{C/C_{rand}}{L/L_{rand}}$, where C_{rand} and L_{rand} denote the clustering coefficient and average shortest path length of equivalent random graphs, respectively. A value of σ greater than 1 indicates small-world network properties. These measures are numerically calculated across multiple simulations, and various statistical measures are similarly computed. Rates of convergence are also noted as important measures of interest.

It emerges that the implementation of R_1 quickly tends toward a saturation of weights (Figure 2, (v) left), as indicated in literature. Furthermore, as weights saturate to 0 it results in the loss of network connections, impacting on overall capacity and computational efficacy. This can be seen by the fact that L blows up to infinity and gives a null result for small-world measure (Figure 2, (iv) left). On the other hand, the implementation of R_2 vastly reduces synaptic oversaturation, preserving a greater distribution of weights (Figure 2, (v) right). This lends evidence to the suggestion that heterosynaptic plasticity may maintain a degree of homeostasis. The "cost" of the network decreases significantly from the randomly initialised graph (Figure 2, (i) right), suggesting that heterosynaptic plasticity optimises efficiency and economy. Finally, this research demonstrates that heterosynaptic plasticity rules tend to stabilise network clustering characteristics, reduce average shortest path length, and therefore produce graphs with σ values greater than 1 – indicating small-world topologies (Figure 2, (ii) to (iv) right).

These findings may have implications for artificial neural networks where there is a need for ever-increasing computational power, while mitigating the constraints of cost and energy consumption. It furthermore may offer insight into the role of heterosynaptic plasticity in various neurological disorders in which brain connectivity studies have demonstrated atypical topologies (e.g. Schizophrenia, Autism Spectrum Disorder, Alzheimer's disease [1, 6, 3]).

References

[1] Pablo Barttfeld, Bruno Wicker, Sebastián Cukier, Silvana Navarta, Sergio Lew, and Mariano Sigman, *A big-world network in asd: dynamical connectivity analysis reflects a deficit in long-range connections and an excess of short-range connections*, Neuropsychologia **49** (2011), no. 2, 254–263. [2] Marina Chistiakova, Nicholas M. Bannon, Maxim Bazhenov, and Maxim Volgushev, *Heterosynaptic Plasticity: Multiple Mechanisms and Multiple Roles*, The Neuroscientist **20** (2014), no. 5, 483–498 (en). [3] Cornelis J Stam, BF Jones, Guido Nolte, Michael Breakspear, and Ph Scheltens, *Small-world networks and functional connectivity in alzheimer's disease*, Cerebral cortex **17** (2007), no. 1, 92–99. [4] Mario Stampanoni Bassi, Ennio Iezzi, Luana Gilio, Diego Centonze, and Fabio Buttari, *Synaptic Plasticity Shapes Brain Connectivity: Implications for Network Topology*, International Journal of Molecular Sciences **20** (2019), no. 24, 6193 (en). [5] Duncan J. Watts and Steven H. Strogatz, *Collective dynamics of 'small-world' networks*, Nature **393** (1998), no. 6684, 440–442 (en). [6] Qingbao Yu, Jing Sui, Srinivas Rachakonda, Hao He, William Gruner, Godfrey Pearlson, Kent A Kiehl, and Vince D Calhoun, *Altered topological properties of functional network connectivity in schizophrenia during resting state: a small-world brain network study*, PloS one **6** (2011), no. 9.