4 Homeostasis

4.1 Synaptic Normalisation

(Video 4.1) Besides STDP, there exist forms of synaptic plasticity that aim at restoring or maintaining a certain activity level in a homeostatic manner. For instance, through homeostatic plasticity, an overly active neuron may weaken its excitatory inputs or an overly silent neuron may strengthen its excitatory inputs. The effect of homeostasis on the activity of a neuron or network of neurons depends strongly on its timescale. Some forms of homeostatic plasticity rescale synapses over hour- or day-long timescales in a multiplicative manner, as shown in rodents after growth or shrinkage of synaptic sizes during experience-dependent synaptic plasticity [1, 2]. We refer to this slow homeostatic plasticity as "synaptic scaling". On the other hand, there is also fast synaptic homeostasis [3], which is able to keep runaway excitation under control [4]. The fast synaptic homeostatic plasticity is most likely heterosynaptic, meaning that the change in one synaptic weight depends on the collective dynamics or states of synapses that are connected to the same postsynaptic target. We call the fast homeostatic plasticity "synaptic normalisation". Until more data becomes available, we assume that synaptic normalisation acts multiplicatively [5], like synaptic scaling. The major advantage of multiplicative synaptic normalisation is that the proportional difference between the smaller and larger weights is conserved. Multiplicative synaptic normalisation therefore does not erase pre-existing memories if it is implemented in a network of neurons. An example of multiplicative synaptic normalisation is the following:

$$w_{j,t+1} = w_{j,t}\left(\frac{W_{\text{tot}}}{\sum_{k=1}^{N} w_{k,t}}\right), \tag{1}$$

Where j and k refer to each synapse onto the neuron, and N is the total number of synapses onto the neuron. The parameter W_{tot} is the value of the total allowed weight onto the neuron, which can be assumed to be flexible over time but we here keep fixed for simplicity. Let us say we wish to have a normalised weight at timestep $t + 1$ in the simulation. The weight $w_{j,t}$ then is the weight just before a normalisation event. After applying equation (20), we obtain the weight just after normalisation, $w_{j,t+1}$, for the timestep $t + 1$. We assume in this case that normalisation events occur periodically, for instance once every second in simulated time.

One can easily see from the equation above that the weight is scaled up or down, depending on whether the sum of the weights is below or above the target value W_{tot}. Each individual weight w_j of course contributes to that sum. However, there is one problem with this equation: it permits instantaneous change of any size to a single weight, which may be very large if the weight is far removed from its target value after scaling. One could argue that such a large instantaneous change is not very realistic in a biological neuron, where a given synapse cannot undergo arbitrarily large changes in a small timeframe. We therefore make a small modification, adding a parameter η_{SN} that represents the proportion of change from the weight prior to scaling. In other words, for η_{SN} the weight will still move towards its value dictated by complete scaling, but will actually only change by for example 20 percent at every scaling event. By modifying the equation above, we obtain

$$w_{j,t+1} = w_{j,t}\left(1 + \eta_{\text{SN}}\left(\frac{W_{\text{tot}}}{\sum_{k=1}^{N} w_{k,t}} - 1\right)\right). \tag{2}$$

The synaptic normalisation rate η_{SN} can be set from any value between 0 and 1, where 1 results in reinstating W_{tot} precisely at each normalisation, equation 19. For our model neuron, let us use $\eta_{\text{SN}} = 0.2$. For practicality, you can implement the normalisation in the neuron simulation to happen every few seconds, depending on how long you simulate your neuron. Synaptic normalisation is also synapse-type specific, occurring separately for all excitatory and all inhibitory weights. Provide the neuron with excitatory inputs from two groups of each 50 spike trains with spike correlations c=0.1 for group 1 and

$c = 0.2$ for group 2, as in the previous section, with STDP, and endow all excitatory synapses with normalisation. Start the weights at 0.1, and use $W_{\text{tot}} = 3$. Also include 30 inhibitory Poisson inputs, with $w_{\text{i}} = 1.0$ and firing rate 10 Hz, but without STDP and normalisation, for simplicity. Normalise the excitatory synapses every second, using equation (21). Run a simulation for 30 seconds. What is happening to the firing rate of the neuron? Is there still competition between the weights of the two input groups? How do the weight distributions of the two groups look at the end of the simulation?
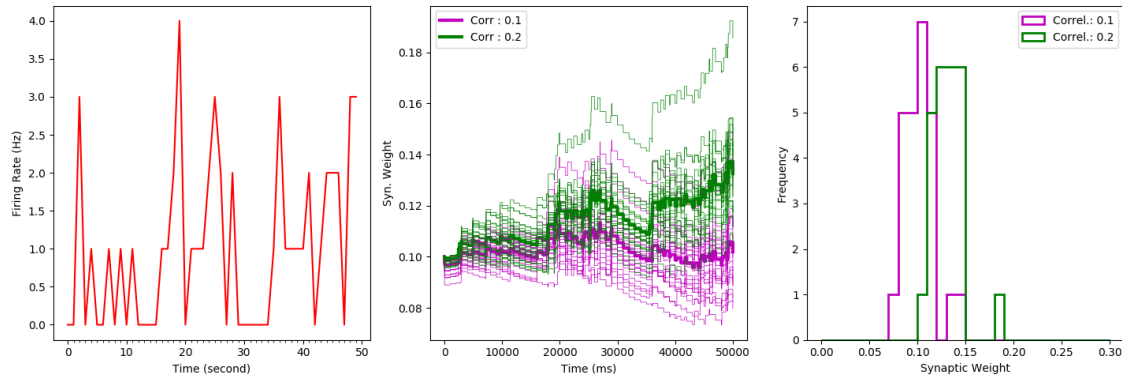

Figure 1: A LIF neuron with two excitatory input groups with STDP and synaptic normalisation in its synapses. The normalisation is applied once every second here. Top left: The LIF neuron is more depolarised over time. Top right: Evolution of the synaptic weights from both input groups. The thin lines show the individual weights, the thick lines the mean weight of the corresponding group. Bottom: The histograms of the weights from each group at the end of the simulation are shown.

References

1. G. G. Turrigiano, K. R. Leslie, N. S. Desai, L. C. Rutherford, and S. B. Nelson, "Activity-dependent scaling of quantal amplitude in neocortical neurons.," *Nature*, vol. 391, no. 6670, pp. 892–896, 1998.

2. T. Keck, G. B. Keller, R. I. Jacobsen, U. T. Eysel, T. Bonhoeffer, and M. Huebener, "Synaptic scaling and homeostatic plasticity in the mouse visual cortex in vivo," *Neuron*, vol. 80, no. 2, pp. 327–334, 2013.

3. J. N. Bourne and K. M. Harris, "Coordination of size and number of excitatory and inhibitory synapses results in a balanced structural plasticity along mature hippocampal CA1 dendrites during LTP," *Hippocampus*, vol. 21, no. 4, pp. 354–373, 2011.

4. F. Zenke, G. Hennequin, and W. Gerstner, "Synaptic Plasticity in Neural Networks Needs Homeostasis with a Fast Rate Detector," *PLoS Computational Biology*, vol. 9, no. 11, 2013.

5. J. Triesch, A. D. Vo, and A. S. Hafner, "Competition for synaptic building blocks shapes synaptic plasticity.," *eLife*, vol. 7, p. e37836, 2018.