Associative memory performance in peripherally-lesioned networks repaired by homeostatic structural plasticity

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Research question

How does repair by structural plasticity affect the recall performance of associative memory stored in an balanced cortical network after focal peripheral lesions?

New model of peripheral lesioning and repair in cortical network

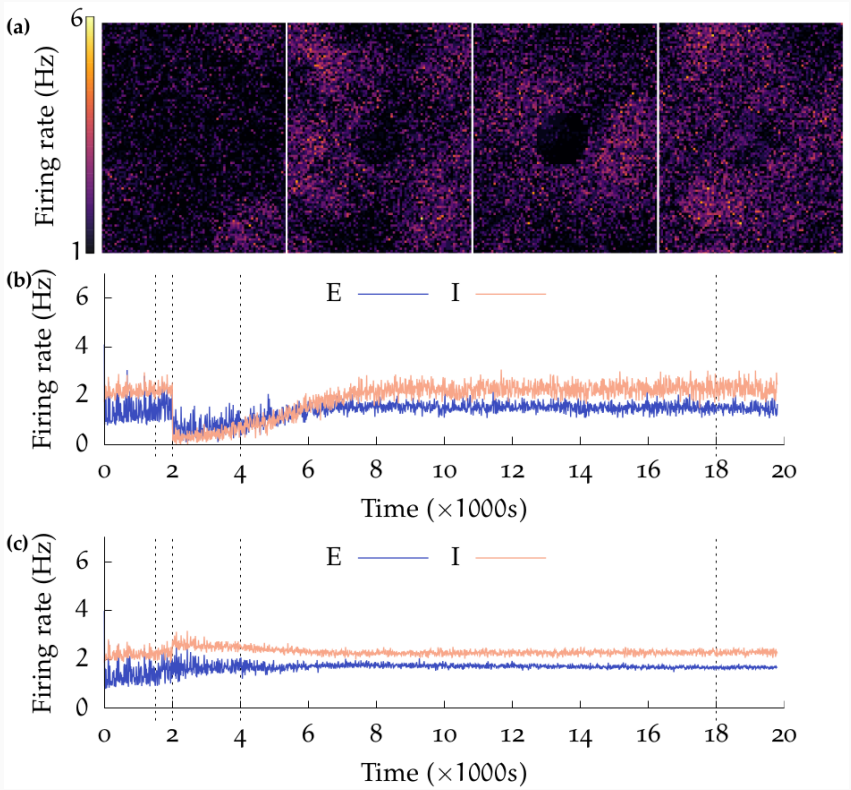

Figure 1: (a) firing rates of the network at different points of time in the simulation. As can be seen in panel 4, activity is restored to deprived neurons in the central Lesion Projection Zone (LPZ). **(b)** mean firing rates of neurons in the centre of the LPZ over time. **(c)** mean firing rates of neurons on the outer periphery of the LPZ over time. The dashed lines in (b) and (c) correspond to the panels in (a).

[1] Manuscript in review, preprint: Sinha, A. *et al.* Growth Rules for the Repair of Asynchronous Irregular Neuronal Networks after Peripheral Lesions. *bioRxiv*. eprint: https://www.biorxiv.org/content/early/2019/10/21/810846.full.pdf. https://www.biorxiv.org/content/early/2019/10/21/810846 (2019)

Model overview

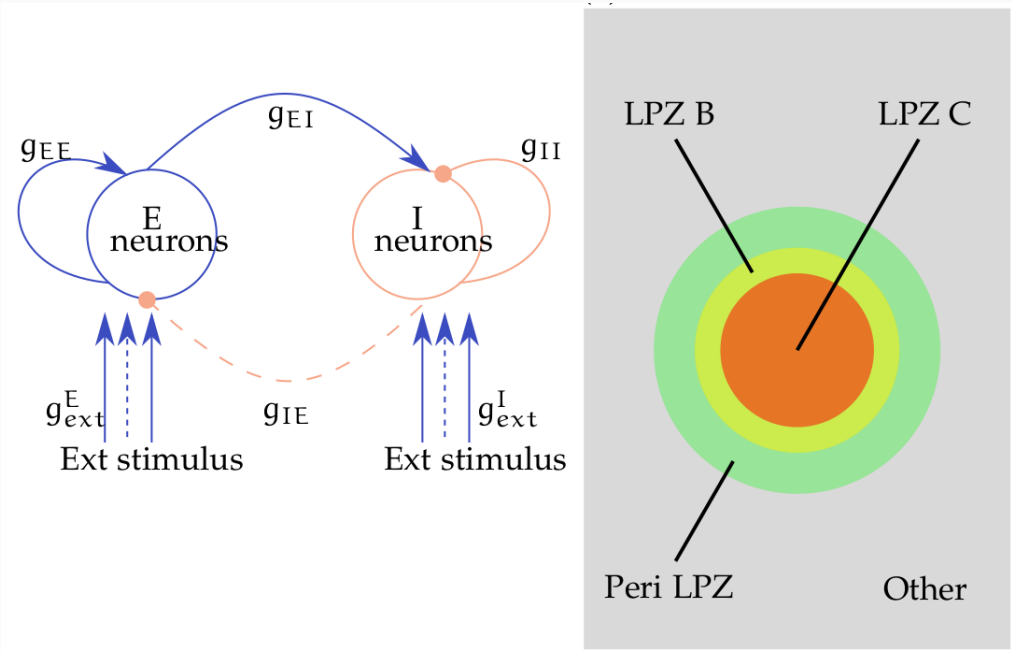

Figure 2: The network consists of excitatory and inhibitory conductance based point neurons connected recurrently, and distributed in a 2D plane. Inhibitory synaptic plasticity[2] modulates the efficacy of IE synapses, while all other synapses are static. Structural plasticity[3] acts on all recurrent synapses in the network. To model peripheral lesion, external input is removed from a set of neurons in the centre of the plane to form the Lesion Projection Zone (LPZ). For analysis, the LPZ is divided into 4 sub-regions, the centre of the LPZ (LPZ C), the inner border (LPZ B), the outer periphery (peri-LPZ), and the remaining neurons.

[2] Vogels, T. P. *et al.* Inhibitory plasticity balances excitation and inhibition in sensory pathways and memory networks. *Science* **334,** 1569–1573. http://www.sciencemag.org/content/334/6062/1569.short (2011)

[3] Butz, M. & van Ooyen, A. A Simple Rule for Dendritic Spine and Axonal Bouton Formation Can Account for Cortical Reorganization after Focal Retinal Lesions. *PLoS Comput Biol* **9,** e1003259 (2013)

Simulation protocol

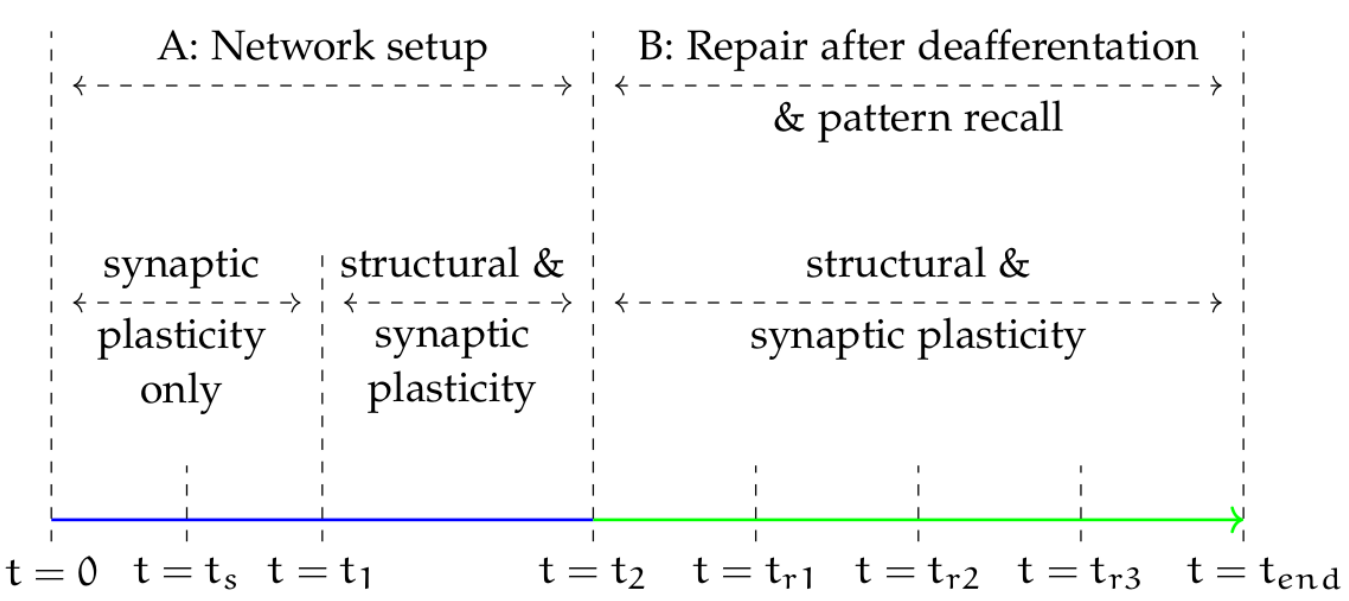

Figure 3: The network is set up with its initial connectivity and allowed to stabilise to its AI regime. Then, at t_s, an associative memory is stored by strengthening the synapses between a randomly selected set of neurons. The network is again allowed to re-stabilise to its AI state. If needed, more patterns are stored in the network in this way. When the last associative memory has been stored and the network returned to its balanced state a snapshot of the network is saved. Then, the stored associative memories are recalled by providing stimulus to a subset of the neurons forming each pattern. The firing rates of the neurons in the associative memory, forming the pattern, and the rest of the neurons of the population, which form the background, allow the calculation of the SNR.

Model predictions I: growth curves for post-synaptic neurites

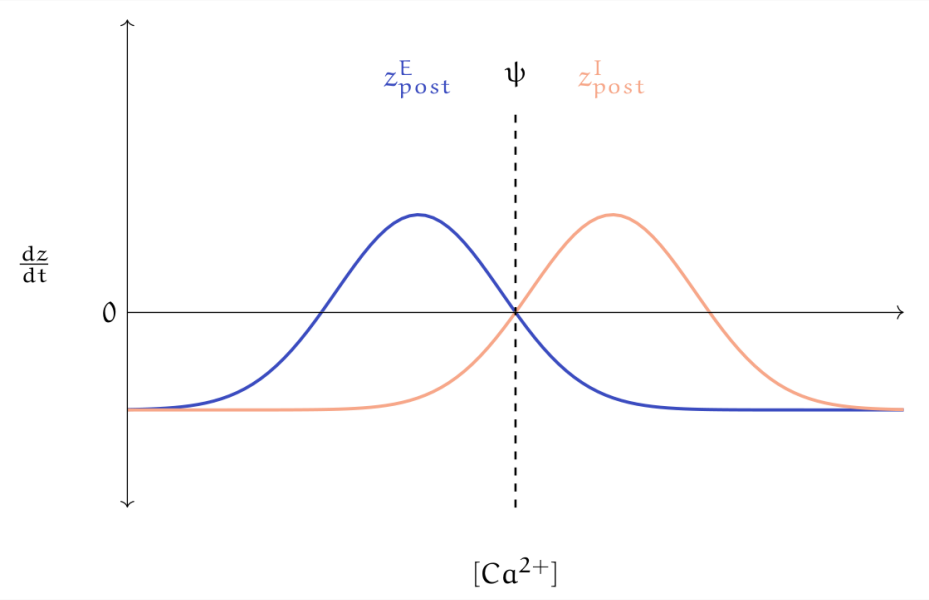

Figure 4: Post-synaptic elements react to changes in activity in opposite ways. dz/dt is the rate of growth for a neurite type, z. Excitatory post synaptic elements z^{E}_{post} are formed when the neuron has less than optimal activity to try and gain more excitatory input but are retracted if the neuron has more activity than it needs to reduce excitatory input. Similarly, if a neuron has more than optimal activity, it forms inhibitory post-synaptic elements z^{I}_{post} to try to gain inhibitory input but will lose them if it has less activity than necessary. Together, even though distilled from network simulations, these growth rules allow single neurons to stabilise their own activity levels.

Model predictions III: growth curves for pre-synaptic neurites

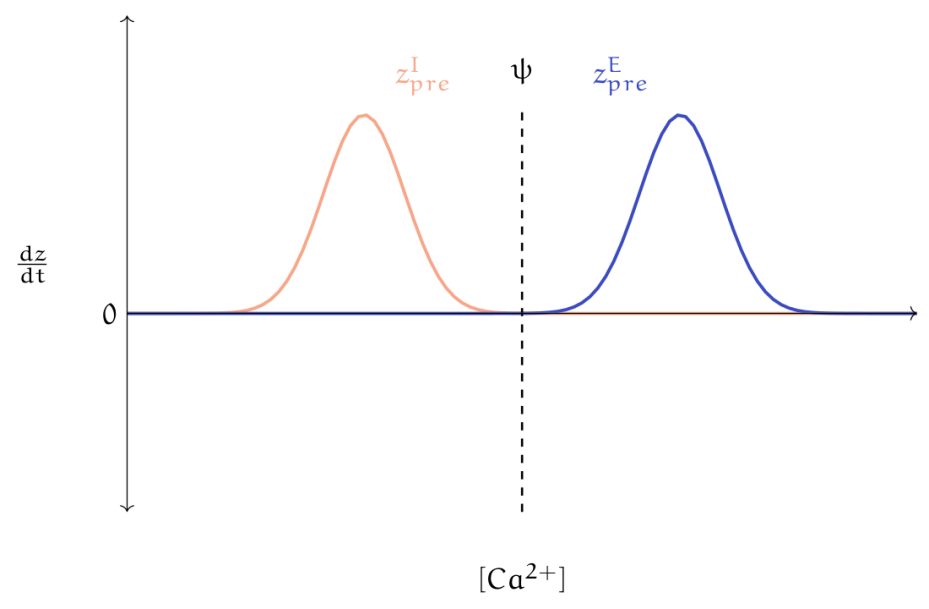

Figure 5: Pre-synaptic elements also react to changes in activity in opposite ways. These were derived by a limited grid search. Excitatory neurons sprout pre-synaptic neurites $z^{E}pre$ when they have more than optimal activity, while inhibitory neurons sprout their inhibitory pre-synaptic neurites z^{I}_{pre} when they have less than optimal activity. In each case, it appears the neurons tries to make the network match its own activity levels, assuming it to be optimal.

Model predictions IV: structural and synaptic both necessary

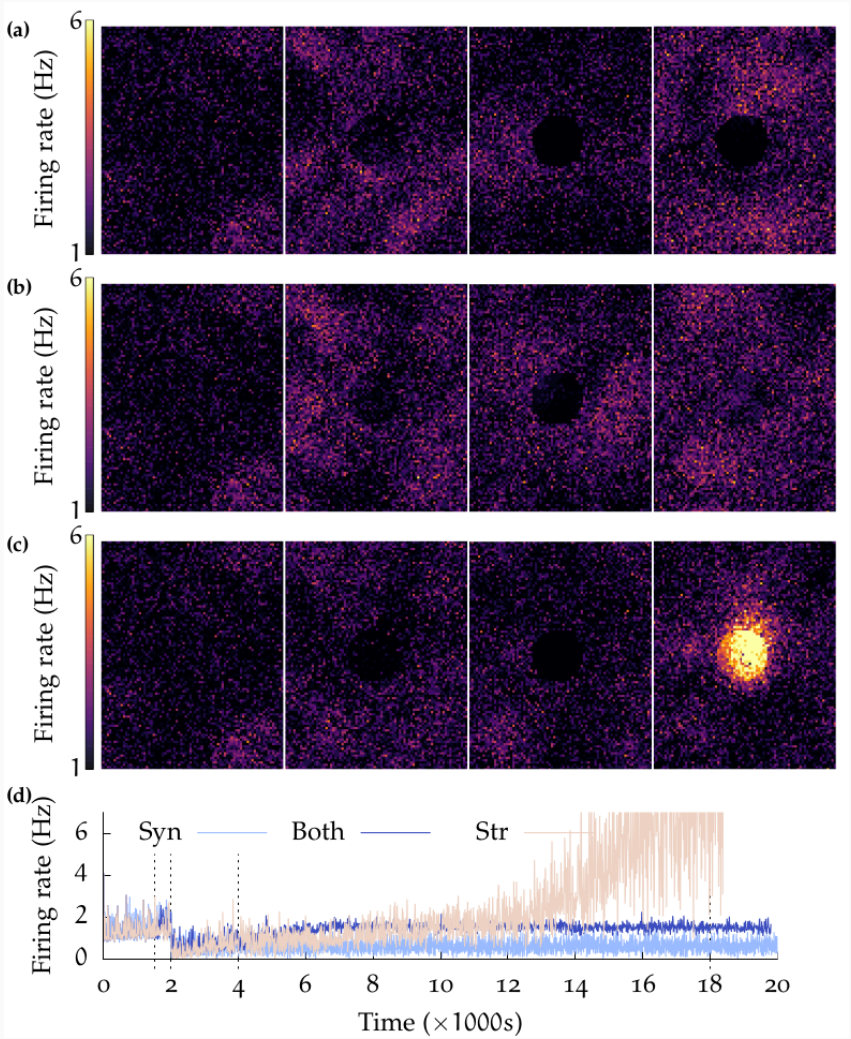

Figure 6: Both structural and synaptic plasticity are necessary for repair. **(a)**: synaptic plasticity only, **(b)** both structural and synaptic plasticity, **(c)** only structural plasticity.

Associative memory performance drops after deafferentation

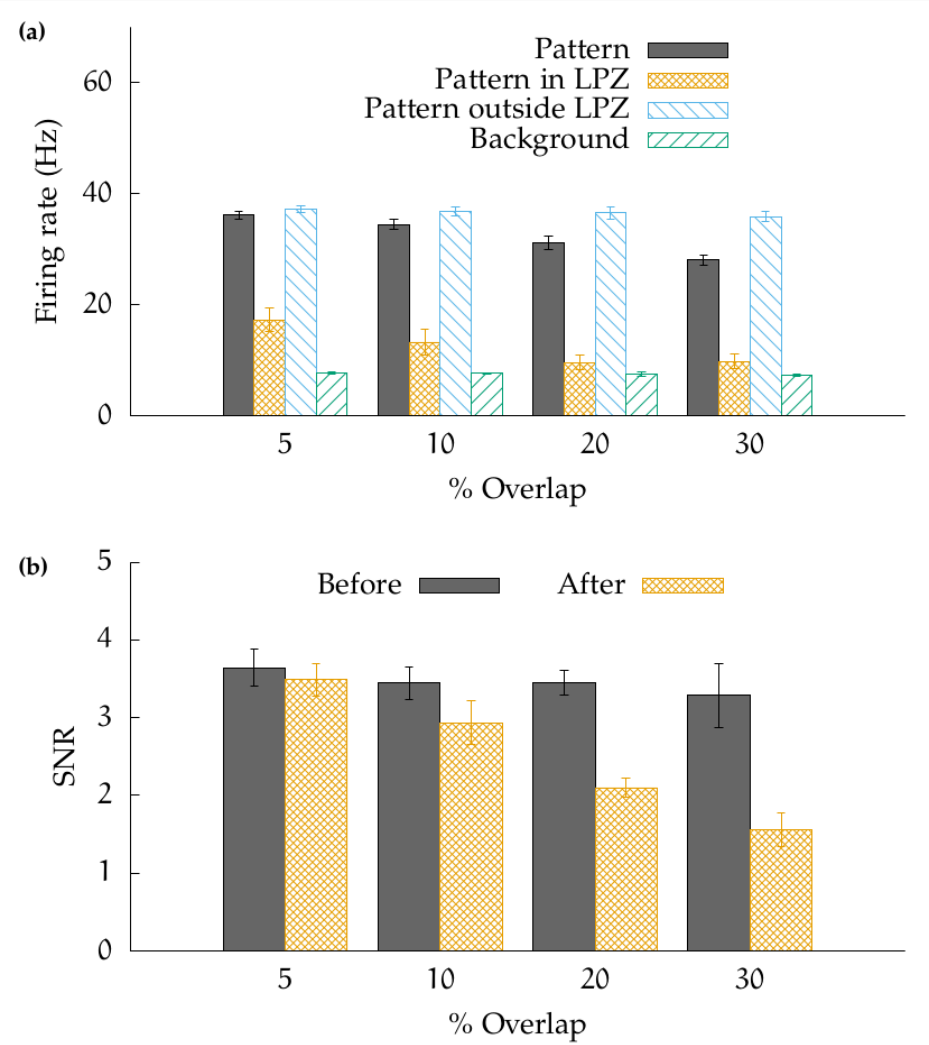

Figure 7: Performance of associative memory recall for varying amounts of the pattern falling in the LPZ (% Overlap). **(a)** population firing rates; **(b)** SNR.

Associative memory performance is not restored by repair

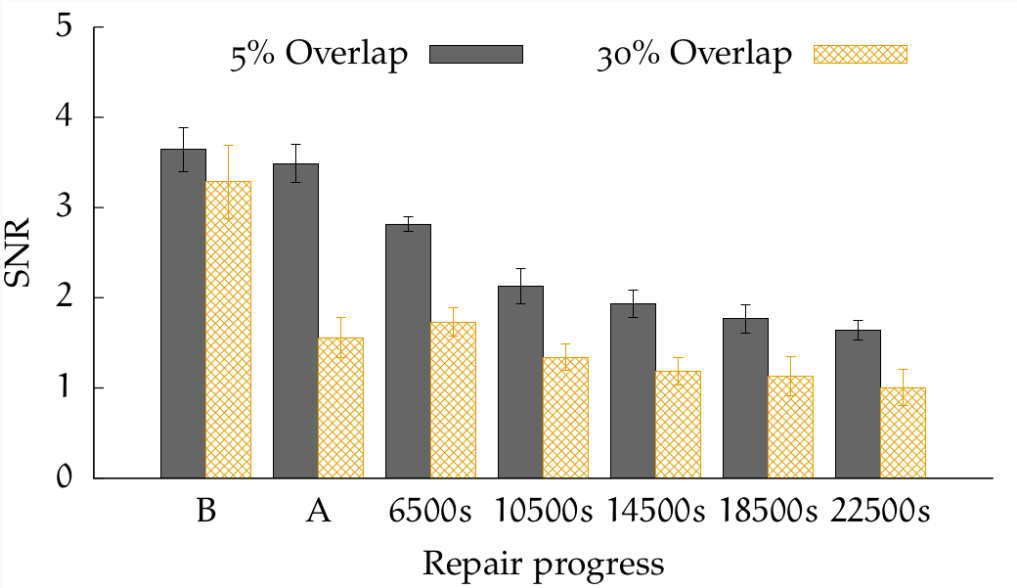

Figure 8: SNR during memory recall before (B) and after (A) deafferentation, and during the repair by structural plasticity for different levels of overlap.

Conclusions

While average activity is restored to the network by structural plasticity after deafferentation, performance of associative memory is not restored.

Open questions for future work

- Experimental validation of peripheral lesioning model[4].
- Should performance be retained after deprivation by peripheral lesions?
- How can the performance be retained/improved?
  - Modulation of the structural plasticity process?
  - Retraining/recall of associative memory during repair?
- If performance is not retained, does the network continue to function as an associative memory store for new memories?

[4] Manuscript in review, preprint: Sinha, A. *et al.* Growth Rules for the Repair of Asynchronous Irregular Neuronal Networks after Peripheral Lesions. *bioRxiv*. eprint: https://www.biorxiv.org/content/early/2019/10/21/810846.full.pdf. https://www.biorxiv.org/content/early/2019/10/21/810846 (2019)

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