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Structural plasticity and memory: Catastrophic forgetting, amnesia, and the spacing effect [1]

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The neurophysiological basis of learning and memory is commonly attributed to the modification of synaptic strengths in neuronal networks. Recent experiments suggest also a major role of structural plasticity including elimination and regeneration of synapses, growth and retraction of dendritic spines, and remodeling of axons and dendrites. Here we develop a simple model of structural plasticity and synaptic consolidation in neural networks and apply it to Willshaw-type models of distributed associative memory [1]. Our model assumes synapses with discrete weights. Synapses with low weights have a high probability of being erased and replaced by novel synapses at other locations. In contrast, synapses with large weights are consolidated and cannot be erased. Analysis and numerical simulations reveal that our model can explain various cognitive phenomena much better than alternative network models employing synaptic plasticity only.

First, we show that networks with low anatomical connectivity employing structural plasticity in coordination with stimulus repetition (e.g., by hippocampal memory replay) can store much more information per synapse by "emulating" high effective memory connectivity close to potential network connectivity. Moreover, such networks suffer to a much lesser degree from catastrophic forgetting than models without structural plasticity if the number of consolidated synapses remains sufficiently low.

Second, we show that structural plasticity and hippocampal replay lead to gradients in effective memory connectivity. This means, neuronal ensembles representing remote memories show a higher degree of interconnectivity than ensembles representing recent memories. Correspondingly, our simulations show that recent memories become more vulnerable to cortical lesions which is similar to Ribot gradients in retrograde amnesia. Previous models of amnesia typically generated Ribot gradients by gradients in total replay time where the M-th memory obtains an 1/M share of replay time, implicitely assuming infinite replay of all memories. In contrast, our model can generate Ribot gradients also with constant replay time per memory. This is more consistent with recent evidence that novel memories are buffered and replayed by the hippocampus for a limited time only.

Third, we show that structural plasticity can easily explain the spacing effect of learning. This means the fact that learning is much more efficient if rehearsal is spread over time compared to rehearsal in a single block. The spacing effect has been reported to be very robust occurring in many explicit and implicit memory tasks in humans and many animals being effective over many time scales from single days to months. For these reasons it has long been speculated about a common underlying mechanism at the cellular level. We propose that structural plasticity is this common mechanism. According to our model, ongoing structural plasticity can reorganize the network during the long time intervals between two rehearsal periods by growing a lot of new synapses at potentially useful locations. Therefore subsequent training can strongly increase effective memory connectivity. In contrast, single block rehearsal can increase effective memory connectivity only slightly above anatomical connectivity.

[1] A.Knoblauch, Proceedings of the 11th Neural Computation and Psychology Workshop, Oxford, 2008

[1] submission to COSYNE 2009, Salt Lake City; send emails to: andreas.knoblauch@honda-ri.de