STDP forms associations between memory traces in networks of spiking neurons

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Supplementary Material

Generation of memory traces

Figure S1: Overview of assembly sizes (blue, green, red) and MRUs (yellow) over 20 simulations with different random seeds.
Figure S2: (A) Emergence of a fourth assembly (brown color) after a new input pattern has been introduced during a 2nd encoding phase (instead of combinations of previously introduced patterns). (B) Mean weight changes of connections from input neurons to the resulting four assemblies (taking only on-channels of the stationary input rate patterns into account) and internal connections within the four assemblies during all phases of the main simulation.
Emergence of associations

Figure S3: Overview of the numbers of PCUs per (blue and green) assembly over 20 simulations with different random seeds.

Figure S4: Scatter plot showing a weak but significant linear correlation between the assembly sizes and the resulting numbers of PCUs per (blue and green) assembly over 20 simulations ($r = 0.45, p < 0.01$).
**Figure S5:** Sources of synaptic inputs to PCUs for the NP stimulus. Grey bars: Firing rates of PCUs with all connections intact. Yellow bars: Firing rates after the internal connections from the assembly of the NP stimulus were disabled. Magenta bars: Firing rates after the input connections were disabled.

**Functional impact of network parameters**

**Figure S6:** HUs as emergent neural code for associations, shown for a network with $\gamma_w = 1.0$ and $\gamma_{exc} = 0.25$ (compare with Figure 5). (A) Response of the network to the first presentation of such a combined pattern during the association phase. (B) After 20 presentations of this combined input pattern during the association phase, a new memory trace represented by 51 HUs encoding the combined input patterns, instead of an overlap between the blue and green assemblies (i.e., no PCUs), emerged.
Figure S7: Impact of various network parameters on the mean assembly size, and the numbers of MRUs, PCUs, and HUs. Mean values were estimated over 10 simulations with different random seeds. Error bars represent the SEM. The * symbols mark the standard parameter values. (A-D) The parameter values of $E_{\text{exc, generic}}$, $I_{\text{exc, generic}}$, and $\gamma := \gamma_{\text{exc}} = \gamma_{\text{w}}$ were varied in logarithmic steps from 25% to 400% relative to the respective standard values. (E-F) The recurrent excitatory connection probability $p_{E \rightarrow E}$ was varied in linear steps from 10% to 90%. More details can be found in the supplemental text below.
In Figure S7A-D, the dependence of the mean assembly size and the numbers of MRUs, PCUs, and HUs on three parameters was investigated in logarithmic steps: $E_{\text{exc,generic}}$ regulates the generic excitability of the excitatory population, $I_{\text{exc,generic}}$ regulates the generic excitability of the inhibitory population, and $\gamma$ scales the relative contributions of the generic excitability of the excitatory population and the initial synaptic weights between $\text{Inp} \to E$ and $E \to E$ connections. We found that the mean assembly size and the number of PCUs roughly peak for values close to the standard value of $\gamma$ and $E_{\text{exc,generic}}$ while they are constantly low for $I_{\text{exc,generic}}$ larger than the standard value and constantly large otherwise. The number of MRUs is negatively correlated with $I_{\text{exc,generic}}$ while being constantly low and largely unaffected by the two other parameters. The number of HUs was found to be negatively correlated with $E_{\text{exc,generic}}$ while being constantly low and largely unaffected by the two other parameters, with a small peak at $\gamma = 70.7 \%$.

In Figure S7E-F, we investigated the impact of the connection probability $p_{E \to E}$ between pairs of excitatory neurons in the recurrent network, using again standard values for all other parameters. The mean assembly size and number of PCUs were found to peak at a connection probability of $p_{E \to E} = 60 \%$ whereas the number of HUs is largely unaffected by the connection probability. The number of MRUs is highly increasing with increasing levels of $p_{E \to E}$. A proper choice of this connection probability is difficult for a model, because its impact on the number of PCUs depends on the size of the neural network model. From the functional perspective it is essential from how many neurons in the same and the associated assembly a neuron in an assembly receives synaptic connections. If this number, which depends on the connection probability and the network size, is too low, few PCUs are likely to emerge. Replacing the biologically more realistic synapse models with short-term plasticity by simpler synapse models with static efficacies did not affect the main results of this study.

A simple calculation shows the following for the subarea CA3 of the hippocampus, which is estimated to consist of 2.83 million pyramidal cells in humans (Andersen et al. 2007) and has an estimated connection probability between pyramidal cells of 0.92 % in rodents (Guzman et al. 2016): An assembly for a memory item in the human MTL was estimated to consist of between 0.2 and 1 % of the pyramidal cells in the MTL (Waydo et al. 2006). Thus with a connection probability of 0.92 %, each neuron in one assembly receives on average synaptic input from 52 to 260 neurons in any other assembly. This suggests that models of different network sizes should have a connection probability that scales this number of presynaptic neurons from another assembly into a comparable range, so that they can contribute significantly to its firing probability. In our small neural network model this number of presynaptic neurons from another assembly had an average value of 12, but in order to achieve that we had to increase the connection probability between excitatory neurons to an unrealistically large value of 50 %. 
Balance between excitation and inhibition

Figure S8: Impact of the scaling factors $\gamma_{\text{exc}}$ and $\gamma_{w}$ on the E/I balance based on membrane potentials at time points (A) after the initialization phase, and directly (B) before and (C) after the formation of associations. Mean values were estimated over 10 simulations with different random seeds. The * symbols mark the standard parameter values. The parameter values were independently varied in logarithmic steps from 25 % to 400 % relative to the respective standard values, as indicated by the black dots. Intermediate grid values were interpolated.

References

