Reconciling forgetting and memory consolidation: simulating the dissociable effects of neuronal noise levels on cortical memory

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Aim

The cortical mechanisms underlying memory acquisition, consolidation, and forgetting remain mostly unexplained. We used a neurobiologically realistic model of neocortical areas to simulate and disentangle the effects of ***neuronal noise*** on such processes.

Background

Noise (as caused by spontaneous neuronal firing) is believed to play an important role in cognitive function: some postulate a contribution to **memory trace decay** (forgetting), yet experimental data indicate that transcranial current stimulation *promotes* episodic memory **consolidation** [1,2]. Does noise induce forgetting or consolidation? Can these different results be reconciled by a unifying model and set of cortical mechanisms?

Methods

Using a neurobiologically realistic Spike-Time Dependent Plasticity rule we trained a deep, neuroanatomically grounded model of sensory, motor, and association areas of the brain (see Fig. 1) to form memory traces (**Cell Assembly circuits, CAs**) linking up "perception" and "action" patterns presented as inputs [3,4]. Two copies of the trained network were then exposed to persistently **high** and **low** noise levels, while **synapses remained plastic.**

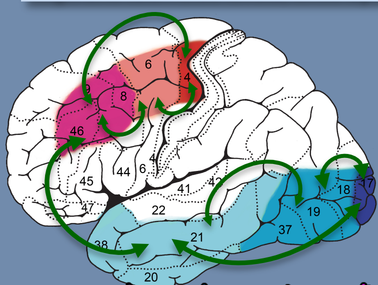
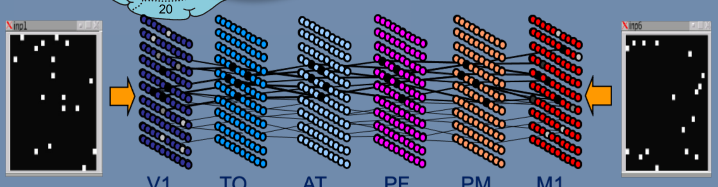


Fig. 1

Left: The modelled brain areas and neuroanatomical links between them.

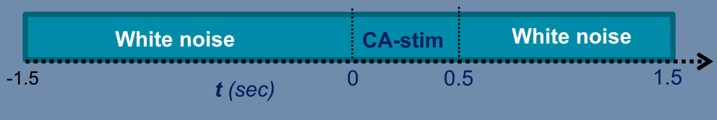
Bottom: Network structure. The colour coding indicates the mapping between brain and model areas.



Forgetting and/or Consolidation (F/C) process

The F/C process consisted of **120 trials** (10 trials per CA circuit, or input stimulus), each trial 3s long, administered under either **high** (=50) or **low** (=5) levels of neuronal noise. We expected that this would lead to different *degrees* of memory-trace decay (forgetting).

FORGETTING / CONSOLIDATION (F/C) trial:



The F/C process was repeated identical on two additional network copies, but **without any CA stimulation** (presenting only **noise**).

Data Collection & Analysis

The strength of the previously learnt memory traces in the different conditions was assessed **(1) prior to**, (**2**) **at the start of,** and **(3) at the end of** the F/C period, by recording network responses (spikes and membrane potentials of all cells) during 12 additional "Testing" trials (= 1 "snapshot") during which **no learning was allowed.**

More precisely, these three network snapshots" were acquired: **(1) at time "zero"**, i.e., immediately after training and prior to the F/C period; **(2) at time "one"**, i.e., after the first 12 F/C trials, and **(3) at time "ten",** i.e., after 120 F/C trials (when each of the 12 learnt input patterns had been presented ten times).

We then used **Morlet wavelet analysis** [5] to assess the presence of oscillatory activity in the model's memory traces during Testing (see Fig. 2). We computed the average spectral power in the 20-to-40 Hz the frequency range in all conditions and ran a repeated-measure ANOVA with factors **Snapshot** (0, 1, 10), **Noise** (Low, High) and **CA-stimulation** (On, Off) on these data (Fig. 3).

Results

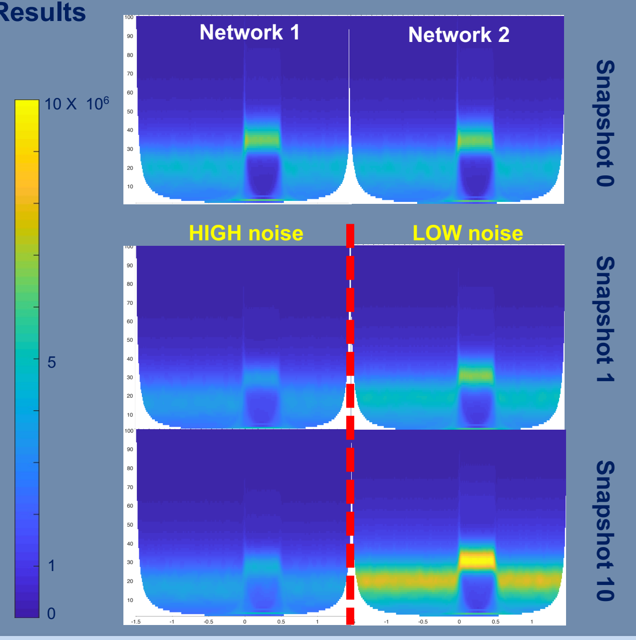


Fig. 2 Simulated induced spectral power at different F/C times.

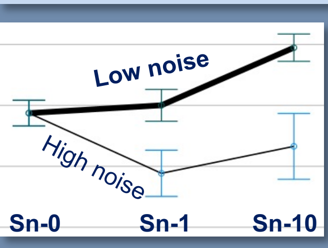


Fig. 3 The ANOVA confirmed a significant **Noise x Snapshot** interaction ($F(2,22)=14.2$, $p=.002498$). There was also a significant CA-stimulation X Snapshot interaction ($F(2,22)=45.4$, $p=.000002$), not shown here.

Conclusions

High noise induced rapid forgetting, but low noise unexpectedly lead to **consolidation of pre-existing memory traces**. These data suggest ***spontaneous re-activation*** (causing strengthening) of existing CA circuits occurring during *low* - but suppressed under *high* - noise levels as a candidate underlying cortical mechanism.

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