# Sequence memory with dynamical synapses

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**Abstract**

We present an attractor model of cortical memory, capable of sequence learning. The network incorporates a dynamical synapse model and is trained using a Hebbian learning rule that operates by redistribution of synaptic efficacy. It performs sequential recall or unordered recall depending on parameters. The model reproduces data from free recall experiments in humans. Memory capacity scales with network size; storing sequences at about 0.18 bits per synapse.

Keywords: sequence learning, free recall, dynamical synapses, synaptic depression, attractor memory

## 1 Introduction

Attractor neural networks as models for cortical memory range from the abstract, such as the Hopfield network, to those incorporating considerable biological realism [4, 7]. Several reports show that key characteristics are not dependent on level of detail, indicating that it is meaningful to study simplified models [3, 9].

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Most versions of attractor memories store static patterns, as fixpoint attractors. Without additional mechanisms, such a memory will reach one recall state and remain there forever. A more complete model of cortical memory should incorporate a way to get out of recall states [13]. This will allow the system to switch between memories; either randomly, according to a learned sequence or in response to external signals.

In the context of symbolic processing, ability to perform such temporal tasks should be built on top of an autoassociative memory. This means that the system retains the ability to represent each memory state individually. Examples of models that do not have this ability are the heteroassociative Hopfield network and pure feedforward networks.

## 2 Model

Our model is a fully connected, single layer, $n$-winner-take-all network with synchronous updating. Neural units are leaky integrators. Synapses are dynamical, depleting part of their resources each time they transmit a signal. The network equations are as follows:

$$\begin{aligned} h_i(t+1) &= (1-\mu_{mem})h_i(t) + \sum_{j=1}^{N} u_{ij} r_{ij}(t) s_j(t) \\ s_i(t) &= \begin{cases} 1 & \text{if } i \in \text{n-argmax}_j \left(h_j(t) + n_j(t)\right) \\ 0 & \text{otherwise} \end{cases} \\ n_i(t) &\in N(0,\sigma) \\ r_{ij}(t+1) &= \left(1 - u_{ij} s_j(t)\right) r_{ij}(t) + \mu_{rec}(1 - r_{ij}(t)) \end{aligned}$$

where $N$ are the number of units in the network, $n$ the number of active units at any one time. Units integrate incoming signals into their internal support value

$h_i$, using a decay parameter $\mu_{mem}$. For the present simulations $\mu_{mem} = \frac{20}{50}$ was chosen, based on one time step in the simulation corresponding to 20 ms of real time, under the assumptions that active units fire once per gamma cycle and that the membrane integration time constant is 50 ms. The vector $s_i$ indicates which $n$ units are currently emitting spikes. The stochastic vector $n_i$ contains Gaussian noise. The synapse connecting the $j$:th unit to the $i$:th expends a fraction $u_{ij}$ of its resources each time it is activated. The expended resources $1 - r_{ij}$ recover at a rate $\mu_{rec}$ (here $\mu_{rec} = 0.025$, corresponding to a recovery time constant of 800 ms) [14].

A Hebbian learning rule is used when training the network, integrating a product of pre- and postsynaptic activity traces. During training, the network activity $s_i$ is "clamped" to teacher signals. In this case $p$ patterns were presented, for one time step each.[1] The learning equations read as follows:

$$
\begin{aligned}
x_j(t+1) &= (1-\mu_{pre})x_j(t) + s_j(t) \\
y_i(t+1) &= (1-\mu_{post})y_i(t) + s_i(t) \\
c_{ij}(t+1) &= \begin{cases} (1-\mu_{learn})c_{ij}(t) + y_i x_j & i \neq j \\ 0 & i = j \end{cases} \\
u_{ij}(t) &= \frac{c_{ij}(t)}{1 + c_{ij}(t)}
\end{aligned}
$$

where $x_j$ and $y_i$ are pre- and postsynaptic activity traces, exponentially decaying as determined by $\mu_{pre}$ and $\mu_{post}$. Coincidences are integrated by $c_{ij}$, optionally with a forgetting rate $\mu_{learn}$. Here $\mu_{learn} = 0$; a non-zero value would create a palimpsest memory [12].

[1] To avoid special cases for the beginning and end of the sequence, training data was presented twice in succession, with plasticity active only during the second presentation.

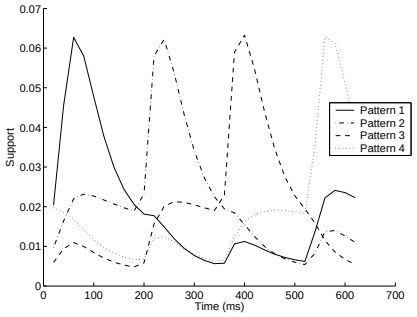


Figure 1: Pattern transitions. The support level of the active pattern declines due to synaptic depression. A fast transition follows.

# 3 Results

## 3.1 Network behaviour

The trained network is principally autoassociative. Started from a random state, it quickly converges to one of the learned patterns. After some time the pattern destabilises. Pattern transitions are sharp, as shown in figure 1. This has also been found in cortical recordings from animals performing sequence tasks [11]. Noise and heteroassociation compete in selecting the next pattern (figure 2). If both $\mu_{pre}$ (generating forward heteroassociation) and $\mu_{post}$ (backward association) are non-zero, sequential recall in either direction is possible as synaptic depression prevents a change of recall direction.

## 3.2 Memory capacity

It is desirable that our model makes efficient use of the information stored in synapses as network size is scaled up. Information content in a single pattern is $I_{pattern} = \log_2 \frac{N!}{(N-n)!} - \log_2 n!$ bits. The limit cycle produced when recall is successful contains $I_{cycle} = pI_{pattern} - \log_2 p \approx pI_{pattern}$ bits. In figure 3 this is plotted in relation to the number of synapses, $N(N-1)$.

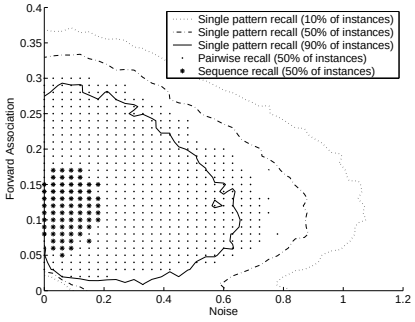


Figure 2: Modes of operation. Network operation breaks down when noise ($\sigma$) and heteroassociation ($\mu_{pre}$) overwhelm autoassociation. In the opposite case patterns never destabilise (lower left). *Contour plots*: probability of 50% of training patterns being produced during a recall episode. *Dots*: same, but counting only patterns followed by the correct successor half of the time. *Stars*: uninterrupted recall of at least half of the training sequence. ($N = 128$, $n = 7$, $p = 50$ (high load), $\mu_{post} = 0$.)

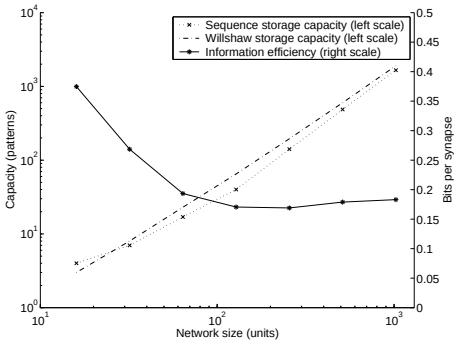


Figure 3: Sequence storage capacity. The network is trained with a sequence of $p$ patterns, then started from a random state and run for $15p$ iterations. Success is when the full sequence is reproduced without error during this time. *Sequence storage capacity*: number of patterns that can be stored while recall is successful at least half of the time. *Willshaw capacity*: loading where we expect all individual patterns to be stable half of the time; $P_2 > 0.5$. *Information efficiency*: based on sequence storage capacity, this is information content $I_{cycle}$ divided by the number of synapses. ($n = \log_2 N$, $\sigma = 0.1$, $\mu_{pre} = 0.1$, $\mu_{post} = 0$.)

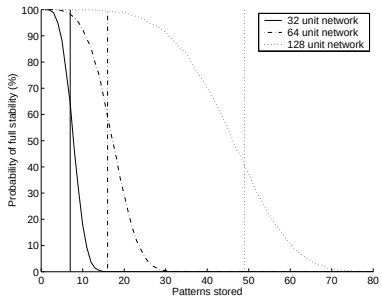


Figure 4: Sequential capacity follows pattern stability. *Curves*: percentage of network instances where all patterns are marginally stable (with no synaptic depression or noise). *Vertical lines*: sequence storage capacity for the respective network size (from figure 3).

Numerical experiments suggest that autoassociative pattern stability predicts success in sequence storage (figure 4). This is to be expected since autoassociation corrects for imperfect heteroassociation when basins of attraction are sufficiently large.

The utilisation parameter nonlinearity $u_{ij} = \frac{c_{ij}}{1+c_{ij}}$ saturates for just a few autoassociative coincidences.[2] We may therefore approximate the $u_{ij}$ as binary, independent stochastic variables and apply the standard analysis of a Willshaw network to autoassociative stability. The probability that a given synapse is non-zero is then $\rho = 1 - (1 - \frac{n(n-1)}{N(N-1)})^p$. If one pattern is fully activated, the probability that a unit outside the pattern will receive enough excitation to destabilise the pattern is $P_0 = \rho^n + n(1-\rho)\rho^{n-1}$. The probability that there is no such unit is $P_1 = (1-P_0)^{N-n}$ and the probability that all patterns are stable is $P_2 = P_1^p$ [1].

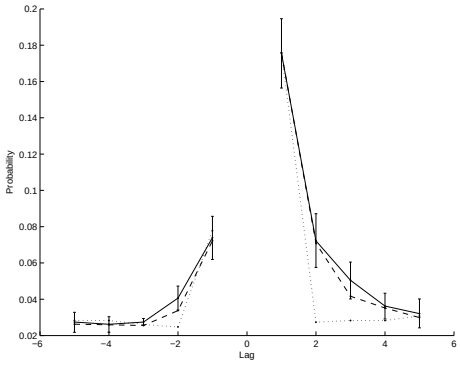


Figure 5: Lag-recency. Solid line is experimental data from the study reported in [10] as analysed in [6]; presentation interval was 1 second, list length 30 items. Dotted line is the response from the basic model. Dashed line is the model response using separate hetero- and autoassociation. Learning parameters were manually tuned to fit data.

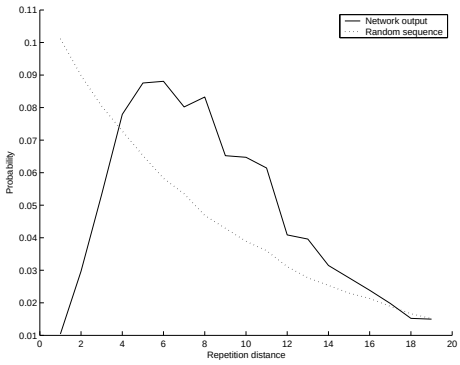


Figure 6: Repetition avoidance due to synaptic depression. Solid line is distribution of repetition intervals in model response, dotted line generated from a random sequence. $p = 11$.

### 3.3 Modelling of free recall experiments

In a free recall task, a participant is asked to recall items from a previously presented list in any order. One effect observed in such experiments is *lag-recency*; participants tend to group items that were close to each other in the original list. Another is repetition avoidance; an item that has been recalled is unlikely to be recalled again for some time [5].

The model reproduces both effects; the former due to the heteroassociative mechanism and the latter due to synaptic depression. As can be seen in figure 5, however, heteroassociation in the basic model raises recall probability only for immediate neighbours. To allow associations that span several presentation intervals, without breaking autoassociation, the forwards and backwards associations were separated out:

$$c_{ij}(t+1) = (1-\mu_l)c_{ij}(t) + \mathsf{f} \cdot s_i x_j + \mathsf{r} \cdot y_i s_j + (1-\mathsf{f}-\mathsf{r})s_i s_j$$

where $\mathsf{f}$ and $\mathsf{r}$ are constants determining the ratios of forward and backward chaining. This can be regarded as a phenomenological model for additional temporal integration mechanisms. While the network thus by simple means reproduces experimental data, it is not intended as a replacement for the more complex models used in this field [5].

Through synaptic depression, the model also implements an approximate "fair scheduling" scheme (figure 6). This is reminiscent of *competitive queueing*, a class of psychological models that have been put forward as an alternative to chaining mechanisms. In this case the mechanism is too weak to produce accurate sequential recall; it only reduces the basins of attraction for recently active patterns [2].

[2]This corresponds to a Willshaw type model. If the contribution to $c_{ij}$ from each coincidence is small, we instead approach the Hopfield regime of linear superposition.

# 4 Discussion

The model presented here performs list recall in random or sequential order. The heteroassociative chaining underlying the latter is robust, though there are more stable models [8]. The core mechanisms of the model are dynamical synapses and learning by redistribution of synaptic efficacy. Synaptic depression enables the system to move out of a pattern, something that would otherwise require a strong external signal. Additional mechanisms, providing cues pointing to the next pattern, would therefore be easily integrated into the present model to form a composite system that can perform more complex serial order tasks.

One easily remedied limitation of the present model is that it does not allow for changing recall pace, which is instead determined by how fast patterns destabilise. Setting synaptic depression such that patterns weaken, but not quite destabilise over time, an external destabilising signal will instead pace recall. One way to implement this in the model would be to temporarily reduce competition, that is increase the number of active units, $n$. Units of the next pattern in line, which is cued but inactive, would then fire alongside those of the active pattern. Since the synapses of the former are at full strength, as opposed to the depressed ones of the active pattern, the latter would be shut down once $n$ returned to normal. In a biological system an equivalent pacing mechanism could be an unspecific signal of either an excitatory or disinhibitory nature. With the addition of such external mechanisms for both serial order and pacing, the present model turns into the core component of a generic sequence processing system.

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