

## Temporal Sequence Compression by a Hippocampal Network Model

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### ABSTRACT

A simple, but biologically plausible, neural network model of hippocampal area CA3 performs temporal sequence compression. That is, the network can recall a sequence in less time than it took to present the sequence during learning. The amount of compression is proportional to the network activity level, which can be adjusted via feedback inhibition.

### 1. INTRODUCTION.

In order to understand the role of the hippocampus in cognition, our laboratory has developed a simplified neural network model of hippocampal area CA3, built with McCulloch-Pitts neurons (McCulloch & Pitts, 1943). The idea behind this network (Levy, 1989) is that the hippocampus learns sequences, which can then be used to solve cognitive mapping problems (O'Keefe & Conway, 1978) like spatial navigation. The model has succeeded in one-trial learning and sequence completion (Minai & Levy, 1993), sequence prediction (Prepscius & Levy, 1994), disambiguation (Minai et al., 1994; Wu et al., 1996), and shortcut-finding (Levy et al., 1995).

While successful in many aspects, networks based on the McCulloch-Pitts model are still too simple to predict actual patterns of neuronal activity. Therefore, we propose new model that is a compromise between the McCulloch-Pitts model and a fully biophysical model. In particular, we incorporate capacitative effects (passive RC decay) into each network element, as well as a timespan for associative modification that exceeds the RC time constant.

As we will show here, the new model displays *temporal compression* by recalling a sequence faster than the time required to present the sequence during learning. The amount of temporal compression is shown to depend on the network activity level.

### 2. METHODS.

Each simulation has two phases, training (unsupervised learning) and testing (recall). During training, a sequence of patterns is presented to the network repeatedly, and synaptic weights are adaptively modified. After the weights have stabilized, the testing phase begins. During testing, the network is prompted with the first pattern of the sequence, and then is allowed to run on its own. If the network has learned the sequence, it will recall a near-approximation of the original (*sequence completion*).

*Network architecture.* The network consists of two layers. The input layer is analogous to the entorhinal cortex and dentate gyrus (EC/DG). Its activity is transmitted with one-to-one connectivity to the main CA3-like layer. The CA3 layer contains 1024 cells, each of which projects excitatory recurrent (feedback) collaterals to only 102 other CA3 cells, reflecting the sparse (10 %) recurrent connectivity in the hippocampus (Amaral et al., 1990). Inhibition is mediated by a feedforward and a feedback interneuron. The feedforward interneuron receives input from all the EC/DG units, and projects to all the CA3 neurons. The feedback interneuron receives input from, and projects back onto, all the CA3 cells.

*Network elements.* The somatic voltage (busline),  $V_j(t)$ , of the  $j^{th}$  cell is updated as follows:

$$dV_j(t)/dt = I_j(t) - V_j(t)/\tau_m$$

where  $\tau_m = 20$  ms is the membrane time-constant and  $I_j(t)$  is the synaptic excitation. When  $V(t)$  crosses the threshold of 0.0003, the cell fires. After firing, a 2 ms absolute refractory period (deadtime) is imposed, and the somatic voltage is decreased by subtracting the threshold.

Because so much of hippocampal inhibition is mediated by  $Cl^-$ -channel activity near the soma (Buhl et al., 1994), we assume that inhibition will be of the shunting type. That is, inhibition will be divided into the cell's excitation, rather than subtracted from it (Furman, 1965). In order to model shunting in a computationally efficient manner, the synaptic current,  $I_j(t)$ , is calculated as:

$$I_j(t) = \frac{excit_j(t)}{excit_j(t) + inhib(t)}$$

The total excitation arriving at neuron  $j$ , is

$$excit_j(t) = x_j(t) + \sum_i c_{ij} w_{ij}(t) z_i(t - \Delta t)$$

where  $x_j(t)$  is the external input,  $c_{ij}$  is a 0/1 variable reflecting the absence/presence of a recurrent connection between cell  $i$  and cell  $j$ ,  $w_{ij}(t)$  is synaptic weight,  $z_i(t)$  is the  $i^{th}$  cell's 0/1 output state, and timestep  $\Delta t = 1$  ms.

The two inhibitory interneurons sample the average activity of each layer and then fire at a rate proportional to this average. For example, letting  $\bar{x}$  and  $\bar{z}$  stand for the average EC/DG and CA3 activities, respectively, the feedforward inhibition is given by  $K_I \bar{x}(t)$ , and feedback inhibition by  $K_R \bar{z}(t)$ , where  $K_I = 100$  and  $K_R = 305$  are scaling constants. The total inhibition is  $inhib(t) = K_I \bar{x}(t) + K_R \bar{z}(t)$ . Running averages  $\bar{x}$  and  $\bar{z}$  are calculated with time-constant of only 2 ms, reflecting the faster response of interneurons compared to CA3 cells (Buzsaki, 1984; Lacaille & Williams, 1990).

*Synaptic Modification Rule.* The network uses a form of unsupervised Hebbian learning which is a modification of Levy (1981). The change in synaptic strength,  $\Delta w_{ij}(t)$ , between presynaptic neuron  $i$  and postsynaptic neuron  $j$  at time  $t$  is given by:

$$\Delta w_{ij}(t) = \epsilon z_j(t) [\bar{z}_i(t - \delta) - w_{ij}(t)]$$

where  $\epsilon = 0.05$  is the learning rate,  $\delta$  is a delay for maximum potentiation,  $z_j(t)$  is the current post-synaptic activity, and  $\bar{z}_i(t)$  is a running average of presynaptic activity. Consistent with the NMDA receptor time-constant (Holmes & Levy, 1990) and other physiological data (Levy & Steward, 1983), we use  $\delta = 5$  ms and a 75 ms time-constant to average the presynaptic activity.

*Network input.* A sequence is composed of  $L = 40$  patterns. For ease of visualization, each pattern is taken to be the activity of 10 adjacent EC/DG units, shifted over by 5 cells from the previous pattern. Each pattern is present continuously for  $T = 50$  ms, making the total sequence duration  $LT = 2000$  ms.

*Sequence completion test.* After training the network for 25 repetitions of the sequence, the testing phase begins. During a sequence-completion test, the EC/DG layer stimulates the CA3 layer with the first pattern of the sequence for 50 ms (the prompt), and then the CA3 layer runs on its own. After the prompt, activity during testing is due solely to recurrent excitation. If the network has learned the sequence, its activity patterns during testing will approximate the sequence of patterns that was present during learning. In such a case, sequence-completion has occurred.

*Measuring temporal compression.* During testing, the network goes through three phases: external prompting, sequence-completion, and repeated recall of the last few patterns. Sequence completion

is thus measured only between the end of the first pattern and the beginning of the final pattern. The compression ratio ( $CR$ ) is a number  $\geq 1$  measuring how much temporal compression the network performs during a sequence completion test. Since the time it would normally take to recall this much of the sequence is  $(L - 2)T$ , the  $CR$  is just the ratio of  $(L - 2)T$  to the measured sequence completion time.

More specifically, define  $\langle t_i \rangle$  to be the average time at which the CA3 cells that *would have* been excited by pattern  $i$  during training (i.e., when externally driven during a learning trial) actually begin firing during recall. Using this notation, the time from the end of the prompt to the beginning of the final pattern is  $\langle t_L \rangle - T$ , and the compression ratio is:

$$CR = \frac{(L - 2)T}{\langle t_L \rangle - T}$$

### 3. RESULTS.

Here we relate the amount of temporal compression to the average activity level of the network. To investigate this relationship, the network is trained at one level of inhibition ( $K_R = 305$ ), and then tested over a range of inhibitory levels, from  $K_R = 20$  to  $K_R = 120$ . (When  $K_R < 20$ , activity is too high to differentiate sequence-related firing from background firing, and when  $K_R > 120$ , activity is too low for sustained firing.)

Results from this experiment are illustrated in Figure 1. It shows CA3 activity at the end of training (Figure 1a) and during two testing trials at the lowest (Figure 1b) and highest (Figure 1c) values of  $K_R$ . Sequence completion is seen in both tests as a diagonal band of activity of neurons that were driven externally during learning. This diagonal band of the first 205 neurons approximates the sequence of activity present during training. Note, however, that sequence completion during testing is faster than sequence presentation during training. For example, although the sequence is presented for 2000 ms during learning, the network recalls the sequence in less than 200 ms in both tests.

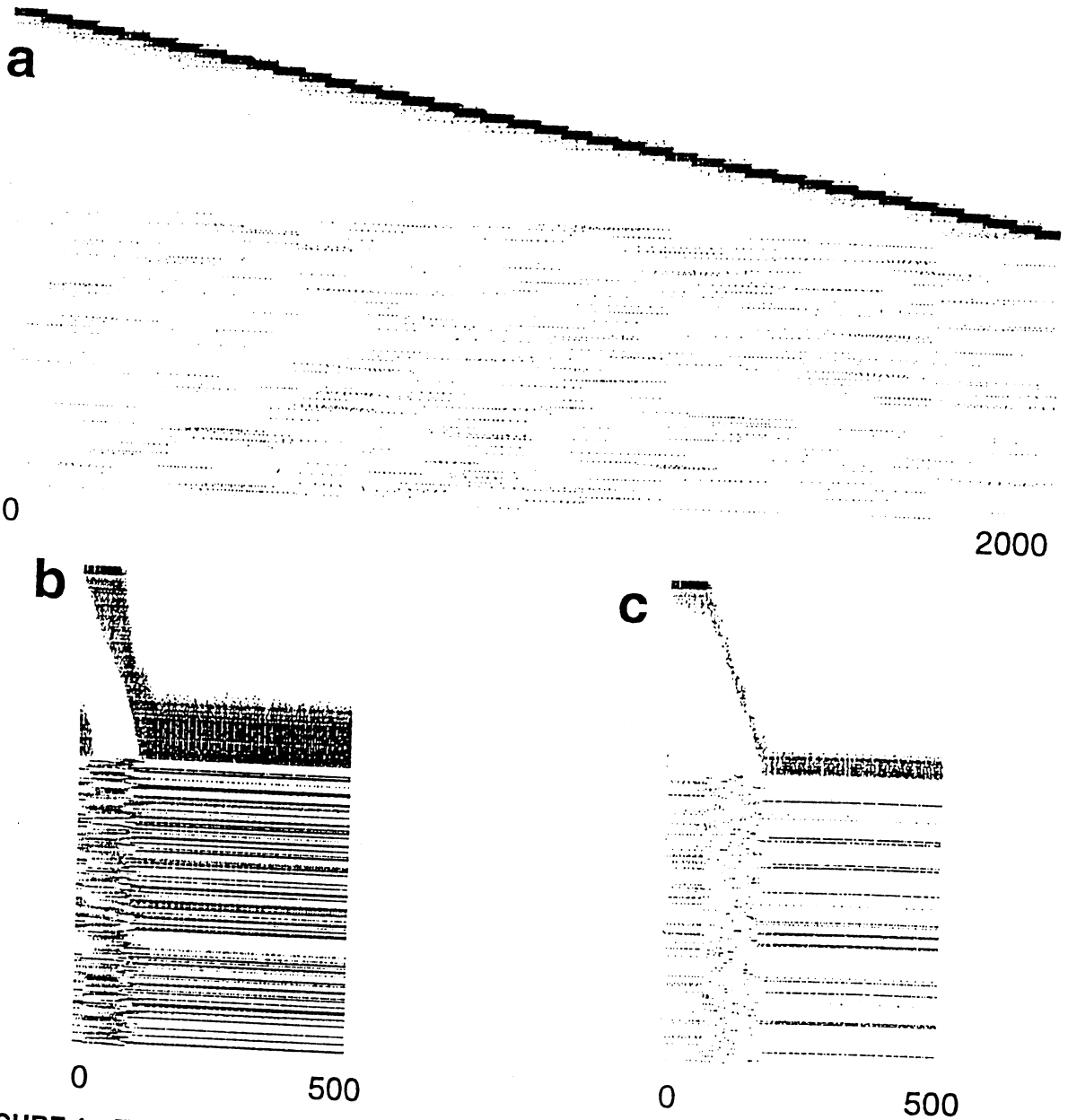
Additionally, there appears to be an inverse relationship between inhibition and compression ratio. For example, when  $K_R = 20$ , the  $CR$  is 14.20 (Figure 1b), and when  $K_R = 120$ , the  $CR$  is 7.01 (Figure 1c). This suggests the possibility a direct relationship between  $m$ , the average network activity during testing, and  $CR$ . To investigate this more quantitatively we combined data from several experiments like the one above, each one with a different random seed. This combined data yields a nonlinear increasing relationship between  $CR$  and  $m$ , which can be fit by the simple function  $CR = 23.189 + 3.814 \ln(m)$  ( $r = 0.94$ , data not shown).

### 4. DISCUSSION.

A biologically plausible model of hippocampal area CA3 is shown here to perform temporal compression. That is, the network can recall a sequence in less time than it took to present the sequence during a learning trial. The amount of temporal compression is a nonlinearly increasing function of the average network activity, and can be controlled by varying the feedback inhibition during testing.

For spatial tasks, the potential utility of temporal compression is apparent. If it took as long to mentally rehearse different possible paths through space as it did to actually navigate them, it would be nearly impossible to plan routes, find shortcuts, or avoid obstacles in a timely manner. For nonspatial tasks, temporal compression may be useful generally for prediction. Human subjects have been shown to exhibit nearly ten-fold temporal compression in a nonspatial sequence-learning task (Sternberg, 1969).

Other investigators have also suggested the possibility of temporal compression in the hippocampus. For example, compression may arise when several patterns of a sequence are compressed into a single phase of the theta rhythm, a 5-10 Hz oscillation of neuronal activity observed in the hippocampus



**FIGURE 1.** *Temporal compression.* (a) 2000 ms of neuronal activity during the last learning trial. Time proceeds from left to right, and the dots in each row represent spiking activity of a single neuron, with neurons 1-512 (out of 1024) shown here. The dark diagonal band of activity results from the external input activating 40 sequential groups of 10 neurons, each for 50 ms. The remaining activity, due to recurrent connections, is characterized by discrete periods of firing that span several patterns. (b) 500 ms of neuronal activity during testing. The network is prompted by the first pattern of the sequence for 50 ms, and then is allowed to run in its own. Here, the strength of the inhibition is  $KR = 20$ , the compression ratio ( $CR$ ) is 14.20, and average activity is 0.09. (c) When tested at higher levels of inhibition ( $KR = 120$ ),  $CR$  falls to 7.04, and average activity decreases to 0.015.

(Skaggs et al., 1996). Although we have not investigated the effect of low-frequency oscillation on temporal compression in this paper, we do note that the network discussed here is capable of high-frequency oscillation ( $> 80$  Hz) when stimulated by random input activity (data not shown).

In this study, activity levels are changed by altering the feedback inhibition. However, in the hippocampus, synaptic transmission is noisy enough (Miles & Wong, 1986) that activity can also be substantially changed by altering the synaptic reliability. In fact, a recent theory of CA3 network function (Hasselmo et al., 1995) proposes that cholinergic modulation dynamically regulates synaptic reliability. Our results appear to remain qualitatively the same when randomly-occurring synaptic failures are included in the model (August & Levy, 1995). However, the effect on temporal compression of activity-dependent changes in the rate of synaptic failure remains to be seen.

Prepscius and Levy (1994) reported that a network of McCulloch-Pitts elements performed temporal compression on a sequence in which each pattern lasted for a single simulation step. Both the strategy and the extent of compression differ from the results reported here, however. For example, here we observe *speed-up* compression, in which the network recalls each pattern for less time than it was present during learning. However, the earlier study observed *jump-ahead* compression, in which the network jumps from the beginning to the end of the sequence without recalling all the intervening patterns. Also observed was *maximal* compression, in which the network simultaneously activates all the neurons participating in the representation of a given sequence. Further, we do not see normal (non-compressed) recall, whereas Prepscius and Levy (1994) did obtain normal recall when the inhibition during training and testing was the same. The first difference may be due to the impossibility of getting speed-up as such when each pattern lasts for only one simulation step. The second difference may be artifactual, due to the difficulty of testing at a high enough  $K_R$  (i.e., the technical problems associated with maintaining low enough activity levels using only 1024 neurons). On the other hand, the compression noted here may be due to the membrane capacitance of the neuronal elements or the relatively long associational time-window of the synaptic modification rule.

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## References

- Amaral, D. G., Ishizuka, N., & Claiborne, B. (1990). Neurons, numbers, and the hippocampal network. In J. Storm-Mathisen, J. Zimmer, & O. P. Ottersen (Eds.), *Understanding the Brain Through the Hippocampus: The Hippocampal Region as a Model for Studying Structure and Function* (pp. 1-11). Elsevier.
- August, D. A. & Levy, W. B. (1995). Incorporating synaptic failures into a CA3 neural network. Abstract 480.9. *Soc. Neurosci. Abstr.*, 1226.
- Buhl, E. H., Halasy, K., & Somogyi, P. (1994). Diverse sources of hippocampal unitary inhibitory postsynaptic potentials and the number of synaptic release sites. *Nature*, 368, 823-828.
- Buzsaki, G. (1984). Feed-forward inhibition in the hippocampal formation. *Prog. Neurobiology*, 22, 131-153.

- Furman, G. G. (1965). Comparison of models for subtractive and shunting lateral-inhibition in receptor-neuron fields. *Kybernetik*, 2, 257-274.
- Hasselmo, M. E., Schnell, E., & Barkai, E. (1995). Dynamics of learning and recall at excitatory recurrent synapses and cholinergic modulation in rat hippocampal region CA3. *J. Neurosci.*, 15(7), 5249-5262.
- Holmes, W. R. & Levy, W. B. (1990). Insights into associative long-term potentiation from computational models of NMDA receptor-mediated calcium influx and intracellular calcium concentration changes. *J. Neurophys.*, 63(5), 1148-1168.
- Lacaille, J. C. & Williams, S. (1990). Membrane properties of interneurons in stratum oriens-alveus of the CA1 region of rat hippocampus in vitro. *Neuroscience*, 36(2), 349-359.
- Levy, W. B. (1989). A computational approach to hippocampal function. In R. D. Hawkins & G. H. Bower (Eds.), *Computational Modeling of Learning in Simple Neural Systems* (pp. 243-305). Orlando, FL: Academic Press.
- Levy, W. B. & Steward, O. (1983). Temporal contiguity requirements for long-term associative potentiation/depression in the hippocampus. *Neuroscience*, 8(4), 791-797.
- Levy, W. B., Wu, X., & Baxter, R. A. (1995). Unification of hippocampal function via computational/encoding considerations. In *Proceedings of the Third Workshop: From Biology to High-Energy Physics. Intl. J. Network Sys.*, volume Suppl., (pp. 71-80).
- McCulloch, W. S. & Pitts, W. (1943). A logical calculus of the ideas immanent in nervous activity. *Bull. Math. Biophys.*, 5, 115-133.
- Miles, R. & Wong, R. K. S. (1986). Excitatory synaptic interactions between CA3 neurones in the guinea-pig hippocampus. *J. Physiol.*, 378, 397-418.
- Minai, A. A., Barrows, G. L., & Levy, W. B. (1994). Disambiguation of pattern sequences with recurrent networks. In *INNS World Congress on Neural Networks*, volume 4, (pp. 176-181)., New Jersey. Lawrence Erlbaum.
- Minai, A. A. & Levy, W. B. (1993). Sequence learning in a single trial. In *INNS World Congress on Neural Networks*, volume 2, (pp. 505-508)., New Jersey. Lawrence Erlbaum.
- O'Keefe, J. & Conway, D. H. (1978). Hippocampal place units in the freely moving rat: Why they fire when they fire. *Exp. Brain Res.*, 31, 573-590.
- Prepscius, C. & Levy, W. B. (1994). Sequence prediction and cognitive mapping by a biologically plausible neural network. In *INNS World Congress on Neural Networks*, volume 4, (pp. 164-169)., New Jersey. Lawrence Erlbaum.
- Skaggs, W. E., McNaughton, B. L., Wilson, M. A., & Barnes, C. A. (1996). Theta phase precession in hippocampal neuronal populations and the compression of temporal sequences. *Hippocampus*, in press.
- Sternberg, S. (1969). Memory-scanning: mental processes revealed by reaction-time experiments. *American Scientist*, 57(4), 421-457.
- Wu, X. B., Baxter, R. A., & Levy, W. B. (1996). Context codes and the effect of noisy learning on a simplified hippocampal CA3 model. *Biol. Cybern.* (in press).

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