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SEQUENCE COMPRESSION BY A HIPPOCAMPAL MODEL: A FUNCTIONAL DISSECTION

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INTRODUCTION

The hippocampus is hypothesized to store memories of experiences during waking hours and then teach the cerebral cortex by broadcasting back this stored information during slow-wave sleep or during moments of wakeful inactivity[1,2,3]. Skaggs and McNaughton[4] identified this rebroadcast in rats that experienced a repetitively sequenced experience. We have presented a hippocampal model of this phenomenon[5,6] which spontaneously rebroadcasts previously learned sequential information. In addition to spontaneous recall, the model also replays the learned sequence information much faster than the original experience, a phenomenon known as temporal compression. The increases produced by the model, ranging from ten to forty-fold faster than the learned rate, are quite comparable to the reported compression observed experimentally by Skaggs and McNaughton[4]. In the model, two parameters are important for controlling the amount of temporal compression. The first is the overall level of activity (e.g., number of cell firings per timestep) and the second is the timespan of the rule governing associative synaptic modification. Increasing either of these parameters can produce more compression.

The hippocampal model, essentially region CA3, contained a moderate amount of biological detail including pyramidal cells of the capacitative, integrate-and-fire type, an inhibitory interneuron with a faster time-constant than the pyramidal cells, and a spectrum of axonal conduction lags on the order of what is seen in the hippocampus. This model encapsulated several different timescales, ranging from the relatively long off-time constant of the activated NMDA receptor (> 100 ms) and the theta-cycle matched duration of individual input patterns (100 ms) all the way down to the short (1-2 ms) time-constant of AMPA class synaptic events and the axonal conduction lags that varied by tenths of milliseconds. The question we now ask is whether all this detail was necessary to simulate temporal sequence compression. That is, can this compression be understood with simpler models?

Here we proceed by simplifying and dissecting the more complicated model; in particular, creating compromises between the integrate-and-fire model and a very simple recurrent McCulloch-Pitts model of area CA3. Specifically, we study four models of increasing levels of complexity. Our goal is to understand the necessity of some of the features included in the integrate-and-fire model.

The four models investigated here use McCulloch-Pitts neurons. That is, the neurons do not have capacitance; they are reset every timestep. Moreover, there is an equivalent time lag (one timestep) of axonal conduction between all connected neurons of this sparsely connected network, rather than a range of axonal conduction delays. Also, in all four of these models, activity is controlled by a simple competetive scheme in which a fixed number of neurons are active at each timestep, rather than being controlled by an interneuron. As always, a local Hebbian associative modification rule spanning at least one timestep is used. The four models are:

- Model 1: The standard McCulloch-Pitts model that this laboratory has been studying as a model of hippocampal function[7,8,9], with an associative modification rule that spans one timestep, and in which individual elements of the input sequence (patterns) also last for just one timestep.
- Model 2: The same model as in (1), but in which input patterns last for multiple timesteps.
- Model 3: The same model as in (1), but with a time-constant in the associative modification rule that extends over several timesteps. This time-constant, denoted τ, is implemented simply as a running averager of presynaptic activity (see, e.g., Levy and Sederberg[10]).
- Model 4: A combination of models (2) and (3), in which input patterns are active for several timesteps and the associative synaptic modification spans several timesteps.

In previous studies, the average network activity level (e.g., the number of cells firing per timestep) was shown to be important in controlling the speed of recall. Therefore, we again study the effect of activity levels on each of the four models described above. In addition, models (2) and (4) allow us to study the effect of input pattern duration on temporal compression. Finally, for models (3) and (4), we investigate the effect of varying the time-constant of associativity, τ, on compression.

Each simulation has two phases, training and testing. During training, the external sequence that drives learning is presented several times to the network, and the recurrent synaptic weights change according to the associative modification rule. After the synaptic weights have stabilized, the testing phase begins. During testing, the network is presented with the first pattern of the sequence as a prompt and is then allowed to run for a period of time without any external input. If learning has been successful, the network will recall an approximate version of the entire sequence on its own.

The input sequence used in these simulations is circular (see Figure 1a). That is, some overlap exists between those cells activated by the first pattern of the sequence and those activated by the (nominal) final pattern. Because of this circularity, the network typically recalls the sequence many times during testing, providing a better opportunity to study the speed and quality of recall. For ease of explicating the simulation, each timestep, Δt, can be thought of as 20 ms.

Figure 1 shows the difference in network firings before and after training for the first and simplest model. During learning, the initially random neuronal firing of the recurrently activated cells (Figure 1a) gives way to a more patterned appearance (Figure 1b), in which individual cells fire in short bursts marking different subsequences analogous to hippocampal place cells.

RESULTS AND DISCUSSION

As shown in Figure 2, model (1) does not perform temporal compression and exhibits poorly selective recall as the average activity level is increased.

Model (2), which includes patterns that last for multiple timesteps, also does not perform temporal compression. In addition, this model has difficulty learning at all when the patterns become too long. For example, when each pattern lasts for 5 timesteps, the network forms an attractor within the sequence, and repeatedly recalls this pattern during testing (data not shown).

In contrast to model (2), model (3) – with its time-spanning associative modification rule – is capable of temporal compression. However, in contrast to the model of August and Levy and as shown in Figure 3a, activity has little effect on the amount of compression, although it again decreases the selectivity of recall. This lower quality of recall makes intuitive sense because, in a situation where each input pattern lasts for only one timestep, compression only comes about by skipping some of the patterns (see Figure 3b). Figure 4 (dashed line) shows the effect of τ, the associative time-constant, on the amount of temporal compression. Increasing τ produces compression. However, this effect appears to plateau.

As implied from the previous model, model (4) also exhibits temporal compression. Moreover, activity increases lead to increased compression, but the additional increase is only 20% when the average activity level is increased from 5% to 50%. Notably, the selectivity of recall at high activity levels is better in this model than model 3 because it is now possible to compress subsequences of individual patterns without skipping them altogether (data not shown). Finally, as shown in Figure 4, higher values of τ produce more compression. Overall, more compression is possible in this model than in model (3), but the five-fold compression shown here is still far from the 20-40-fold compression observed experimentally and observed in the August and Levy model.

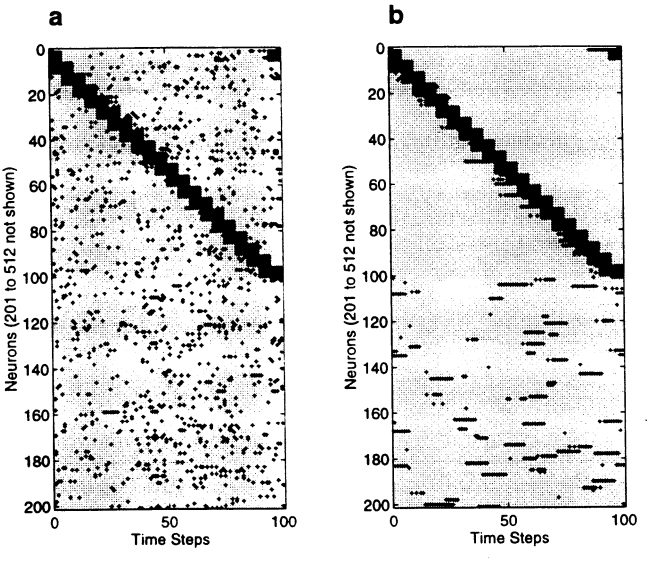

Figure 1: *Network states before and after training.* (a) This rastergram depicts the state of the network in model (1) in response to external input before synaptic modification. Each large dot is a firing event, thus each row is the firing pattern of one cell over time. Think of the network in two parts (although there is no such explicit distinction). The first part contains cells directly activated by the external input; the second part contains the neurons that are only fired, in this example, by recurrent excitation. Here, the first part corresponds to the first 100 cells and the second part is the remaining 900 cells of the 1000-cell network. The diagonal band of large dots starting in the upper left corner of each figure is the firing of those cells that are directly activated by the 20-pattern input sequence. The remaining, recurrently-activated cells, appear to fire randomly in Fig. (a). Note that to save space, only the first 200 cells of the network are shown. (b) After 100 presentations of the sequence, the recurrent synaptic weights have changed and stabilized, cells no longer fire randomly, instead, cells fire over brief periods of time spanning a few consecutive input patterns analogous to hippocampal place cells.

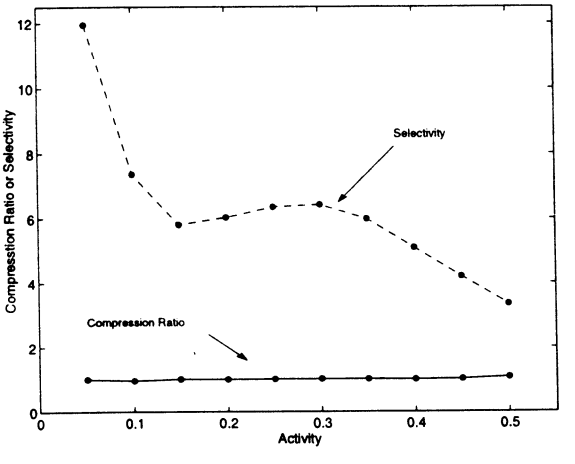

Figure 2: *Effect of activity on model (1).* As activity levels during recall increase, the selectivity decreases, while the compression ratio remains the same. The compression ratio, CR, is the ratio of the original to the recalled sequence duration. As shown here, $CR \approx 1$ no matter what activity level at which the network is tested. The selectivity is the ratio of the highest similarity value at any timepoint during recall to the average of all the other similarity values at that time, where similarity is measured by a normalized product of the network state matrix at the beginning and ending of training.

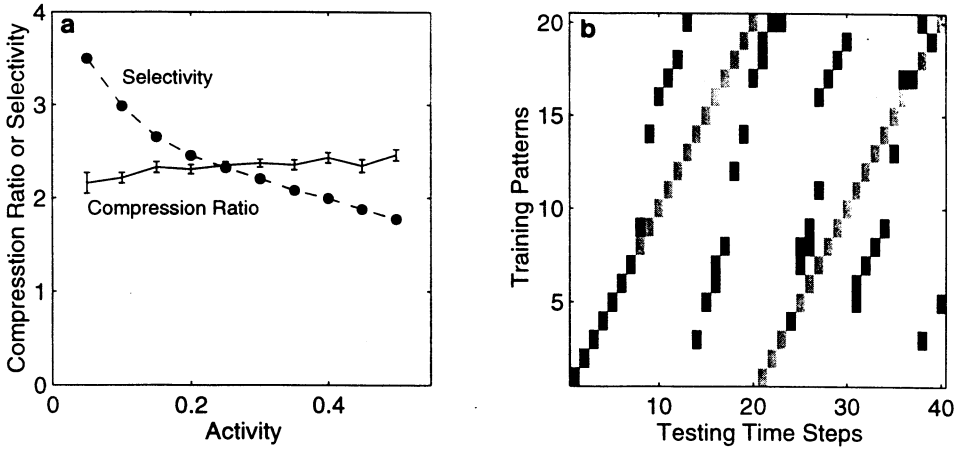

Figure 3: *Effect of activity on recall for Model (3).* (a) Unlike the two previous models, this model does compress the sequence by about 2-fold. Increasing the average activity level during testing from 5% to 50% increases compression somewhat, but still decreases the selectivity of recall substantially. (b) This panel shows the "winners" as a function of time during recall. The winner at any given timestep is the pattern, of the last learning trial, that has the highest similarity value to the network state as one timestep during testing. Thus, this depression represents a crude decoding of the CA3 network state. Here, the winners are shown in black and the original sequence is shown for comparison in gray. Compression is apparent based on the increased slope of the sequence of winners compared to the original sequence. However, also apparent, during each recall of the sequence, is the tendency of this network to skip patterns which lowers the selectivity of recall. Here, the network is running at 5% activity and $\tau = 100$ ms.

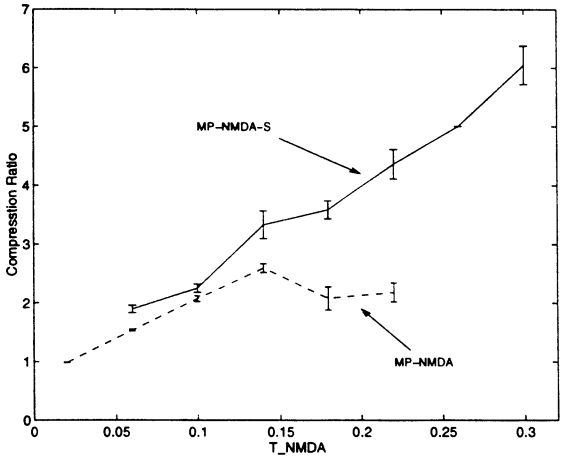

Figure 4: *Increasing τ increases temporal compression.* The compression ratio increases with τ, the time-constant of the running averager of presynaptic activity in the synaptic modification rule. Shown here are simulations using model (3) in which input patterns last for 20 ms (dashed line), and model (4) in which input patterns last for 100 ms (solid line). Two effects are apparent. First, as τ increases, so does compression. There is a range, for relatively low values of τ, for which increasing the pattern duration makes little difference in the amount of compression. However, using longer duration patterns allows us to extend the range over which τ can increase CR to produce up to 5-fold compression. Increasing τ much beyond the range of values illustrated here leads to failure of recall.

In conclusion, temporal compression can be achieved with a McCulloch-Pitts based recurrent network that lacks the complexity of a multi-timescale model with integrate-and-fire, capacitative neurons. A model studied previously by our laboratory similar to model (1) does perform a type of compression called jump-ahead, in which the network simply skips from early in the sequence to the final pattern during recall[11]. Although model (1) studied here can reproduce this behavior (data not shown), it is not our primary interest here because it does not resemble the type of sequence compression observed experimentally by Skaggs and McNaughton[4].

In our simulations, the crucial feature for obtaining compression is the presence of some time-spanning capability in the rule governing associative synaptic modification. However, although simpler models qualitatively work, they fail to produce quantitative predictions of compression seen in behaving animals and are not robust to parameter settings. The earlier, more physiological model, with its more detailed appreciation of the timescales of various biophysical events, is much more appropriate for predicting quantitative aspects of temporal compression.

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REFERENCES

1. Buzsaki, G., Horvath, Z, Urioste, R., Hetke, J., and Wise, K., 1992, High-frequency network oscillation in the hippocampus, *Science* 256:1025-1027.
2. Pavlides, C. and Winson, J., 1989, Influences of hippocampal place cell firing in the awake state on the activity of these cells during subsequent sleep episodes, *J. Neurosci.* 9(8):2907-2918.
3. Wilson, M.A. and McNaughton, B.L., 1994, Reactivation of hippocampal ensemble memories during sleep, *Science* 265:676-679.
4. Skaggs, W.E. and McNaughton, B.L., 1996, Replay of neuronal firing sequences in rat hippocampus during sleep following spatial experience, *Science* 271:1870-1873.
5. August, D.A., 1996, Sequence learing with an integrate-and-fire neural network model of hippocampal area CA3. Dissertation thesis.
6. August, D.A. and Levy, W.B, 1997, Spontaneous replay of temporally compressed sequences by a hippocampal network model, in: Computational Neuroscience: Trends in Research 1997, J.M. Bower, ed., Plenum, New York, 231-236.
7. Levy, W.B, Wu, X.B., and Baxter, R.A., 1995, Unification of hippocampal function via computational/encoding considerations, *Int. J. Neural Sys.* 6 (Supp.):71-80.
8. Levy, W.B and Wu, X.B., 1996, The relationship of local context codes to sequence length memory capacity, *Network* 7:371-384.
9. Wu, X.B., Baxter, R.A., and Levy, W.B, 1996, Context codes and the effect of noisy learning on a simplified CA3 model, *Biol. Cyber.* 74:159-165.
10. Levy, W.B and Sederberg, P.B., 1997, A neural network model of hippocampally mediated trace conditioning, *IEEE International Conference on Neural Networks* I-372-376.
11. Prepscius, C. and Levy, W.B, 1994, Sequence prediction and cognitive mapping by a biologically plausible neural network, *INNS World Congress on Neural Networks* IV-164-169.