

A PDP Model of Spacing Effects in Memory

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Introduction

The spacing effect has been well known by psychology since the 19th century yet there is still no consensus on the causes of the effect. Pavlik and Anderson (2003) proposed a math model explanation in which the strength of a memory at the time of practice determines the forgetting rate for what is learned from that practice. This math model captures various results in the spaced practice literature by proposing that forgetting is greater for what is learned when a memory is already strong. The question remains, however, given what we know about the neurophysiology of the hippocampus and the properties of cells in this area, why do these equations provide a good summary of the data?

One of the most elucidating studies is Scharf et al. (2002). In this experiment, tetanic stimulation was applied at 100 Hz 4 times to hippocampal slices in vitro. These 4 tetanic pulses were applied with either a spacing of 20 seconds between pulses or 5 minutes between pulses. Additionally, some slices in both conditions were pretreated with the protein synthesis inhibitor anisomycin. In both conditions, anisomycin blocked LTP and activity returned to near baseline within 3 hours. In contrast, without anisomycin, significant LTP was observed for both narrowly and widely spaced stimulation. As the spacing effect would suggest LTP was much greater in the spaced condition even though initial EPSC as a percent of baseline was only about 20% higher in the spaced condition.

A Neural Model

A PDP model was constructed to explain what might underlie the Pavlik and Anderson (2003) equations. This 200-unit auto-associator model describes how the hippocampus CA3 region may cause the behavioral spacing effect due to properties of neurons similar to those found by Scharf et al. (2002). The central mechanisms of the model depend on the links between units. Each link has two types of link strength: short-term potentiation strength (STP), and long-term potentiation strength (LTP). STP strength is determined according to the delta rule using basic principals of autoassociator learning. For any practice, the STP link strength increases by the weight change matrix multiplied by the learning rate. In addition, for every time step (equal to the average time of a trial, about 10 seconds) STP links decay. The exponential decay rates for STP links are randomly sampled from an exponential distribution with mean 0.038. By having this distribution of decay rates, the observed forgetting of the network more closely matches the power function forgetting observed behaviorally. This

agrees with work by R. Anderson (1997) showing that an average of different exponential decay functions is fit by a power function. Further, this is likely to be more accurate than characterizing weight decay with a single rate constant. Current work has described at least 3 kinds of potentiation and 1 type of depression, each with different rates of return to baseline. (e.g. Schulz and Fitzgibbons, 1997). This STP mechanism in the model is strong enough to capture basic short-term recency and frequency memory effects.

LTP link strength is determined according to the delta rule using the weight change matrix and a matrix of learning rates. Using a matrix of learning rates means that each link has its own individual learning rate. The initial value for each link's learning rate is equal to a single parameter, the base LTP learning rate. LTP link strength does not decay.

Every time an LTP link is strengthened, the LTP learning rate for that link is decreased by a factor of the LTP learning that occurred at that link. This mechanism accounts for link "fatigue" and may be explained as due to the expenditure of LTP related chemicals, which require a period to recover following link strengthening. Consequently, there is also a parameter that describes exponential decay recovery of LTP link learning rates to the original base LTP link learning rate. By restricting long-term gain from closely spaced practice this LTP learning fatigue mechanism results in the spacing effect observed behaviorally.

The model captured all the main effects and interactions in the data from Pavlik and Anderson (2003) (an overall *RMSD* of 0.0694 and an r^2 of 0.930). The success of this model using neurally plausible mechanisms suggests that the properties of neurons are sufficient to capture high-level behavioral effects of spaced practice.

References

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