A Source of Individual Variation

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Abstract. Even though behavioral experiments are usually conducted in a manner that minimizes variance between subjects, such variance is unavoidable – even when working with a homogenous population. A first step in producing a quantitative understanding of what causes this variation is a biologically-based neural network model that reproduces the observed individual differences. Here we show that a simple model of the hippocampus can reproduce the histogram of learned performance in a homogenous population. The task and data being fit is transitive inference that is learned by college students. Individual differences arise from initial connectivity differences not neuronal state initializations.

Introduction. Variation is a property of all biological processes, and it seems an unavoidable observation in cognitive psychology.

A simple hypothesis is that individual variation between similar subjects arises from variation in the underlying neurobiology. However, which aspect of the neurobiology produces the individual variation being studied is a much more difficult question. Here we advance an answer to this question for learning that requires the hippocampus.

Because our simple model of the hippocampus reproduces the learning curves and variability of several 'hippocampal' tasks, we decided to fit human learned performance for one of these tasks, specifically transitive inference. To summarize the results, the individual variation between simulations arises from variation of initial connectivity at the beginning of training. On the other hand, the variation of each such simulation across multiple trials arises from variation of initial neuron activities at the start of such test trials.

The model. The model used here is a hippocampal model of region CA3 (see Fig. 1 and, e.g. Levy 1996 for more details). The input layer corresponds to a combination of the entorhinal cortex and dentate gyrus. To make the system's operation as transparent as possible, decoding is performed by similarity comparisons rather than a CA1-subiculum-entorhinal decoding system. The CA3 model is a sparsely (10%) interconnected feedback network of 1024 neurons where all direct connections are excitatory and the network elements are McCulloch-Pitts neurons. There is an interneuron mediating feedforward inhibition and one mediating feedback inhibition. Inhibition is of the divisive form, but the system is not purely competitive because of a slight delay. Synaptic modification develops over training. The process controlling synaptic modification is a local, self-adaptive postsynaptic rule that includes both potentiation and depression aspects (Levy & Steward 1979). The network computations are all local and are contained in three equations: spatial summation adjusted by inhibition; threshold to fire or not; and local Hebbian synaptic modification (see e.g. Levy 1996 for details).

The transitive inference problem. In the transitive inference problem, five atomic stimuli (e.g. A, B, C, D, and E) are used. When A and B are together, A is the right answer (A>B). When B and C are together, B is the right answer (B>C) and so on. Thus, subjects and networks are taught A>B, B>C, C>D, and D>E. The critical test of TI after learning these four pairs is the response to B?D. Of course, the correct answer is B>D, and rats require a hippocampus to solve this problem (see e.g. Dusek and Eichenbaum, 1997).

Learning and decoding. As part of our hypothesis of the computation performed by the hippocampus (Levy 1989), we have cast this problem as sequence prediction (Levy & Wu, 19997; Wu & Levy, 1998; Wu et al. 1998). That is, sequences of stimulus-decision/response-reinforcement are presented to the network.

For the TI problem, a staged learning paradigm (Dusek & Eichenbaum 1997) is used. Thus, there are eight sequences to learn in this case:

(AB)(AB)(AB)aaa+++, (AB)(AB)(AB)bbb- - -,

(BC)(BC)(BC)bbb+++, (BC)(BC)(BC)ccc- - -,

(CD)(CD)(CD)ccc+++, (CD)(CD)(CD)ddd- - -,

(DE)(DE)(DE)ddd+++, and (DE)(DE)(DE)eee- - -.

During testing, the network is initialized and given the novel stimulus pair (BD) for three time steps all the while turning on a fraction of the "+" input code. The recurrent network responds, generating its own sequence of cell firings. We decode this sequence to see what the network predicts (e.g., b or d) will produce the desirable + event. Decoding uses the cosine comparison to find the most similar states to the defined external inputs; that is, we compare each state during testing to the previously defined input firing patterns.

Our computational modeling of the transverse patterning problem has been described many times (e.g., Levy 1996; Levy et al., 1996; Wu et al., 1997, 1998; Shon et al., 2000).

Variability. By design, only two randomizations occur for the simulations here: 1) the connectivity of each simulation, which produces the variability across simulations, and 2) the initial firing pattern, Z(0), at the beginning of each training trial.

Training and testing are always initialized in the same way. That is, when we systematically vary the probability of this random neuronal activity at time step zero, testing uses the same random process (i.e., valued Bernoulli process independent across neurons) as was used for generating Z(0) in the corresponding training trials.

Results. First we point out that initial state randomization can improve performance.

Figure 2 plots network performance on the transverse patterning problem as a function of Z(0) randomness. As the probability of CA3 neurons firing at initialization Z(0) varies from 0.0 to 0.5, the frequency of good performance reaches a maximum at 0.1, the desired activity level (Shon et al., in press; Wu & Levy 1999). This result motivates us to fix the Z(0) activity at 0.1 in the transitive inference simulations.

Figure 3 shows the distribution comparing the number of correct transitivity responses (b for the BD test) that occurred in the transitive inference problem; here we are comparing seven human subjects and fourteen simulations of the network model. Despite small sample sizes, the human experiment and the computer model show remarkably similar response distributions. To match the experimental data, again the activity level and synaptic modification constant are adjusted. The small chi square value (0,75;d.f. = 6) corresponds to very similar histograms.

Discussion. We have shown that a simple neural network model can produce individual variation similar to human subjects. Because the neuronal updating is deterministic, there are only two possible sources that can produce this variation; it either comes from the randomization of cell firing at the beginning of each learning trial or it comes from the initially specified random connectivity of the network.

Interestingly, the best performance is achieved at full initial state randomness (Fig. 2). This is consistent with, and extends, our previous findings that a moderate level of randomness (Wu & Levy, 1999) and activity fluctuation (Levy & Wu, 2000, Smith et al., 2000) during encoding helps learning. That is, the activity fluctuations of the more biological, recurrently inhibited model correlate with much better performance compared to the competitive model (Levy & Wu 2000). Furthermore, the best performance does not occur at the zero gradient where activity fluctuations are minimized (Smith et al., 2000). This also suggests that, once performance is substantially above zero, increasing the variability of initial states at the beginning of each training trial reduces the variance across simulations. (See also Sullivan & Levy, this conference, for another kind of random fluctuation that improves performance.)

Even so, it is this trial-to-trial activity randomization that causes variations when testing a single simulation many times: that is, initial state randomization is what produces the simulations that neither make all correct, nor all

incorrect, decisions (see Fig. 3). Thus, we conclude that connectivity randomization is the cause of variation between simulations. By way of confirmation, when the initial state randomizations are made constant across different simulations (but still random from trial to trial), then the same variation across simulations results. Thus, the connectivity randomization is causing the variation across simulations.

No problem is more central to the goals of psychological science than to understand the cause of individual variation, why one person is cognitively different than another. Although when asked in terms of the biological basis of cognition such a question seems hopelessly complex by virtue of possible answers, a computational model leads us to a simple, specific hypothesis – at least for the variation in learning, in a homogenous population when learning depends on the hippocampus.

Acknowledgments

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Figure Legends

Figure 1. The hippocampal model. Entorhinal and dentate inputs are collapsed together; the excitatory recurrent connectivity is sparse but dominates over the external inputs in determining total neuronal firing. Inhibition is both feedforward and feedback.

Figure 2. Learned performance is affected by the Z(0) randomization in the transverse patterning problem. The frequency of good performance is plotted as a function of randomness at time step zero where a fixed number of neurons (here 102) are activated. That is, we always have 102 neurons turned on at time step zero for the data shown in this figure. For example, if we have 92 randomly chosen firing neurons, then another 10 fixed neurons are always turned on for compensation. The randomness is defined as (n*p-nf)/(n-nf) where n is the network size, p is the desired percent activity (0.1 here), and nf is the number of fixed neurons for compensation. Thus, when nf=102, the randomness is zero (0). When nf=0, we have the case of full randomness. Note that the best performance is achieved at the full randomness.

Figure 3. Frequency distribution comparing of the number of correct transitivity (BD) responses in the inference test for seven human subjects and fourteen simulations of the computer model. Despite small sample sizes, the human experiment and the computer model show remarkably similar response distributions. For both the human experiment and the computer simulations, there were four premise pairs of five atomic stimuli learned via the staged training paradigm. For the human experiment, the stimuli were five distinct Kanji characters described elsewhere (experiment 1 of Greene et al., 2001). For the computer model, orthogonal blocks of neurons coded each stimulus of a pair.

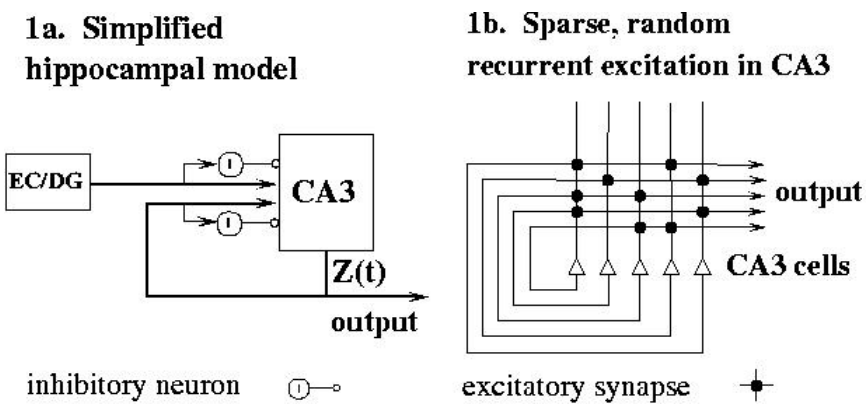

Figure 1.

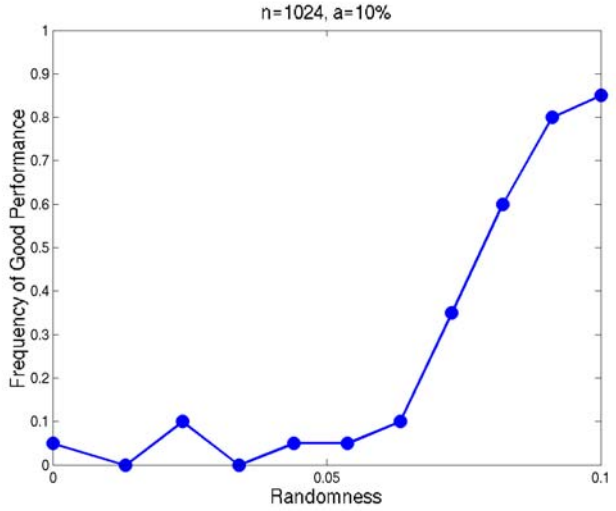

Figure 2.

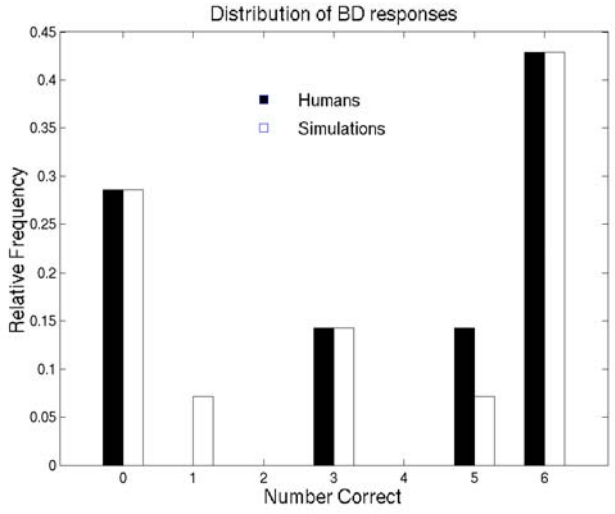

Figure 3.