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 mainly 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. 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.

The model. The model used here is a hippocampal model of region CA3 (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 and Smith et al., 2000 for details). 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). Network parameters were: $K_r = 0.055$; $K_i = 0.016$; $K_0 = 0.13$; initial weights were 0.4; threshold was 0.5; and synaptic modification rate was 0.02.

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).

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 (see, e.g., Shon et al., 2002).

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. Figure 1 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, the activity level and synaptic modification constant were adjusted but all simulations are parameterized identically. 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. This is consistent with, and extends, our previous findings that a full initial state of randomness (Shon et al., 2002) helps learning. It is this trial-to-trial initial state 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. 1). However, because initial state randomization is negatively correlated with variation once average performance is above 50%, we conclude that connectivity randomization is the main 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 individual networks 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.

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Figure Legends

Figure 1. 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.

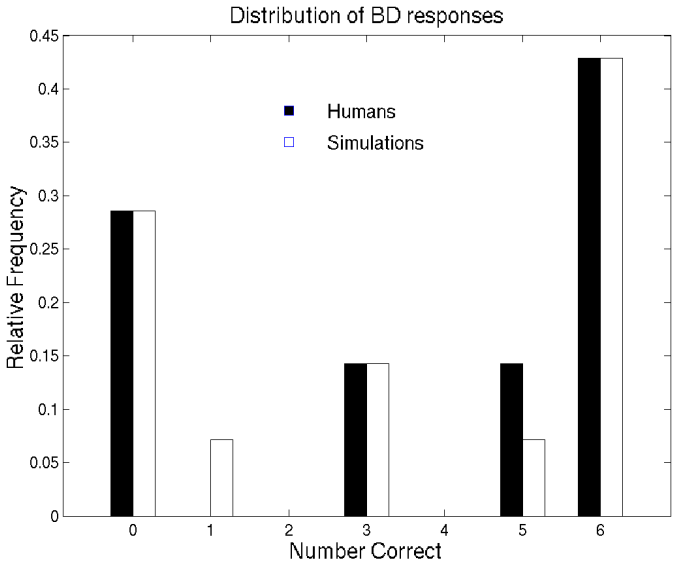
Distribution of BD responses
Humans
Simulations
Relative Frequency
Number Correct
0.45
0.4
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
0
1
2
3
4
5
6

Figure 1.