Assessing the Chaotic Nature of Neural Networks

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Abstract

During the course of brain development there exists a brief period of exuberant synaptic formation followed by an equally impressive pruning and strengthening of surviving synapses that eventually form the basis of the adult brain. The root factor in shaping this process is the information we receive from the world in this critical period of development, the effects of which last a life-time. As we learn neural circuits are fine tuned to accomplish meaningful tasks be they locomotion, communication or any other of the myriad of cognitive facilities we possess. In the process uncertainty gives way to programmatic perfection, those shaky first steps become effortless strides. With this in mind I hypothesized that learning reduces chaos in neural circuits. Below is an initial proof of concept for accessing the chaotic nature of neural circuits.

1   Introduction

1.1   Synaptic pruning and learning

It is well documented that in the course of development of the human nervous systems there is an early explosion of the presence of synapses, that peeks around two years after birth, and that over the course of childhood are pruned to reach the adult state [1, 2]. This pruning coincides with the acquisition of many skills. As such it is rather simple than to conclude that the exuberance of synapses primes the brain to absorb, process, and retain any and all information it is exposed to. Indeed, this is extremely evident when one considers language. There are an approximately 7,000 known living human languages in the world [3], and without exception every child, at birth, has the capability of flawlessly mastering the languages they are exposed to, correlatively, anyone who took a foreign language class in high school or college can attest to the increased difficulty of doing so after the synapses of our language centers have been pruned. More rigorously changes in synaptic density have been observed by electron microscopy under various conditions of sensory deprivation in an assortment of model organisms, however; results are not entirely concordant [4, 5, 6, 7].

Concomitant with synaptic pruning is the strengthening of those synapses not pruned. It is believed that those synapses not pruned are the ones encoding useful information, and therefore their influence on network activity is increased, not only by eliminating those synapses not contributing to information processing in a proactive manner, but in enhancing their individual contribution. This entire phenomenon, that is a drastic pruning of synapses with concomitant strengthening of those synapses surviving pruning, has been shown in computational models of neural networks with synaptic dynamics modeled by spike time dependent plasticity (STDP) [8, 9, 10]. STDP of course, has been well established as a basis for learning.

1.2 Chaos, small differences magnify

The field of chaos theory dates back at least to the 1880's when Henri Poincaré was studying the three-body problem [11], but really took off 50 years ago in part due to the advent of computers that could effortlessly churn through thousands of computations, and in part due to the good fortune of Edward Lorenz. Lorenz serendipitously discovered that, when given enough time, very small changes in the initial conditions to his simple three state deterministic model of weather resulted in drastically different end results [12].

This sensitivity to initial conditions is mathematically characterized by calculating the maximal Lyapunov exponent. This exponent, defined in Equation 1,describes how the time evolution of the relationship between two infinitesimally close points (initial separation δZ_0) in the state-space of a system. A positive Lyapunov exponent signifies they diverge exponentially, making it impossible to make long term predictions of the nature of the system. A negative Lyapunov exponent signifies points converge, and a zero Lyapunov exponent signifies some periodic nature. As such a system is said to be chaotic if the maximal Lyapunov exponent of the system is positive.

$$\lambda = \lim_{t \to \infty} \lim_{\delta Z_0 \to 0} \ln \left(\frac{|\delta Z(t)|}{|\delta Z_0|} \right) \tag{1}$$

2 Methods

2.1 Forward modeling of network activity

Individual neurons were modeled as Hodgkin-Huxley neurons [13] with synaptic inputs modeled by (NEED INFORMATION). A geometric graph with 30 vertices (neurons) and 107 edges (synapses) was then generated such that the network exhibited scale free connectivity. Synaptic weights were randomly assigned from a uniform distribution ranging from -1 to 1. Synaptic delays were based on the distance between vertices and ranged between 52 and 232 ms ($\bar{x} = 124.4672$, $\sigma = 49.1118$). A 20 ms depolarizing current pulse of 10 uA was then delivered to a single neuron to set the network in motion. Network dynamics were calculated for 600 ms at a 50 kHz sampling rate using an Euler integrator.
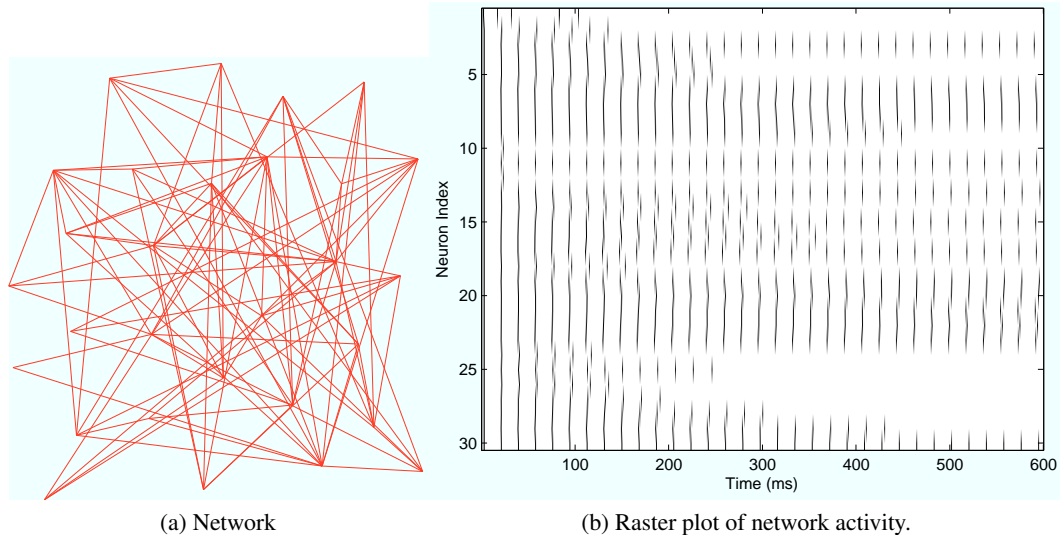

(a) Network   (b) Raster plot of network activity.

Figure 1: Forward modeling of network activity.

2

2.2 Determination of Lyapunov exponents

Since it is rarely possible to analytically solve for the maximal Lyapunov exponent of a dynamic system various numerical methods have been developed [14], here I use the "standard" method. The state of the network, X, is fully described by an n-dimensional state-space vector. The dynamics of the network as described above is formulated as a series of n-dimensional coupled differential equations, S, that we evaluate at discrete times using the Euler integration method (Equation (5)). At each time step the Jacobian, J, of the system is evaluated along the fiducial trajectory. The Jacobian is then multiplied by the δZ matrix from the previous time step, the initial δZ matrix being the identity matrix.

$$X = \begin{bmatrix} x_1 & x_2 & \ldots & x_n \end{bmatrix}^T \tag{2}$$

$$S(X) = \begin{bmatrix} f_1(X) & f_2(X) & \ldots & f_n(X) \end{bmatrix}^T \tag{3}$$

$$= \begin{bmatrix} \frac{dx_1}{dt}(X) & \frac{dx_2}{dt}(X) & \ldots & \frac{dx_n(X)}{dt} \end{bmatrix}^T \tag{4}$$

$$X(t+1) = X(t) + S(X(t))\Delta t \tag{5}$$

$$J(t) = \begin{bmatrix} \frac{df_1}{dx_1}(X(t)) & \frac{df_1}{dx_2}(X(t)) & \ldots & \frac{df_1}{dx_n}(X(t)) \\ \frac{df_2}{dx_1}(X(t)) & \frac{df_2}{dx_2}(X(t)) & \ldots & \frac{df_2}{dx_n}(X(t)) \\ \vdots & \vdots & \ddots & \vdots \\ \frac{df_n}{dx_1}(X(t)) & \frac{df_n}{dx_2}(X(t)) & \ldots & \frac{df_n}{dx_n}(X(t)) \end{bmatrix} \tag{6}$$

$$\delta Z(t+1) = (I + J(t)\Delta t)\delta Z(t) \tag{7}$$

This is done at each time step for τ time steps, at which point the Gram-Schmidt reorthonormalization (GSR) method is applied to the δZ matrix. The norms, $\|\hat{z}_i\|^{(m)}$, of the vectors of the matrix prior to normalization during the m-th GSR step are stored for later use, and the normalized matrix, $\delta\hat{Z}$, is used as the new δZ matrix. This process is carried out for the entire stretch of the simulation, after which the Lyapunov exponents are determined as described in Equation (11). Ideally this process should be carried out until the exponents converge.

$$\delta Z = \begin{bmatrix} z_1 & z_2 & \ldots & z_n \end{bmatrix} \tag{8}$$

$$\delta\hat{Z} = \begin{bmatrix} \frac{\hat{z}_1}{\|\hat{z}_1\|} & \frac{\hat{z}_2}{\|\hat{z}_2\|} & \ldots & \frac{\hat{z}_n}{\|\hat{z}_n\|} \end{bmatrix} \tag{9}$$

$$\hat{z}_i = z_i - \sum_{j=1}^{i-1} \frac{<z_i, \hat{z}_j>}{<\hat{z}_j, \hat{z}_j>} \hat{z}_j \tag{10}$$

$$\lambda_i = \lim_{r \to \infty} \sum_{m=1}^{r} \frac{\ln(\|\hat{z}_i\|^{(m)})}{r\tau} \tag{11}$$

Reorthonormalization is carried out for three purposes. First, for the practical purpose of minimizing the chance of numerical overflow. Second, to reduce the chance of underestimating the Lyapunov exponents which may occur due to reasons of space folding, and lastly to maintain an accurate description of the tangent space of the system [15].

Delays are implemented by the addition of state variables to the system [16], which consequently increases the size of the Jacobian, such that old values of critical state variables are carried along for the minimum number of time steps required. This is accomplished as described in Equation (12), where η represents some integer number of time step delays.

$$\frac{dx_i^{(\eta)}}{dt} = \frac{1}{\Delta t} \left(x_i^{(\eta-1)} - x_i^{(\eta)} \right) \tag{12}$$

Code that implements the simulation of network dynamics as well as the calculation of the Lyapunov exponents was written in a combination of Matlab and C++ using the CUDA library to enable parallelization on GPGPUs.

3

3 Results

Due to time constraints the Lyapunov exponents were calculated only for the system described earlier. The Lyapunov exponents of the system quickly converge within 300 ms, and the system is determined to be chaotic (see Figure 2).
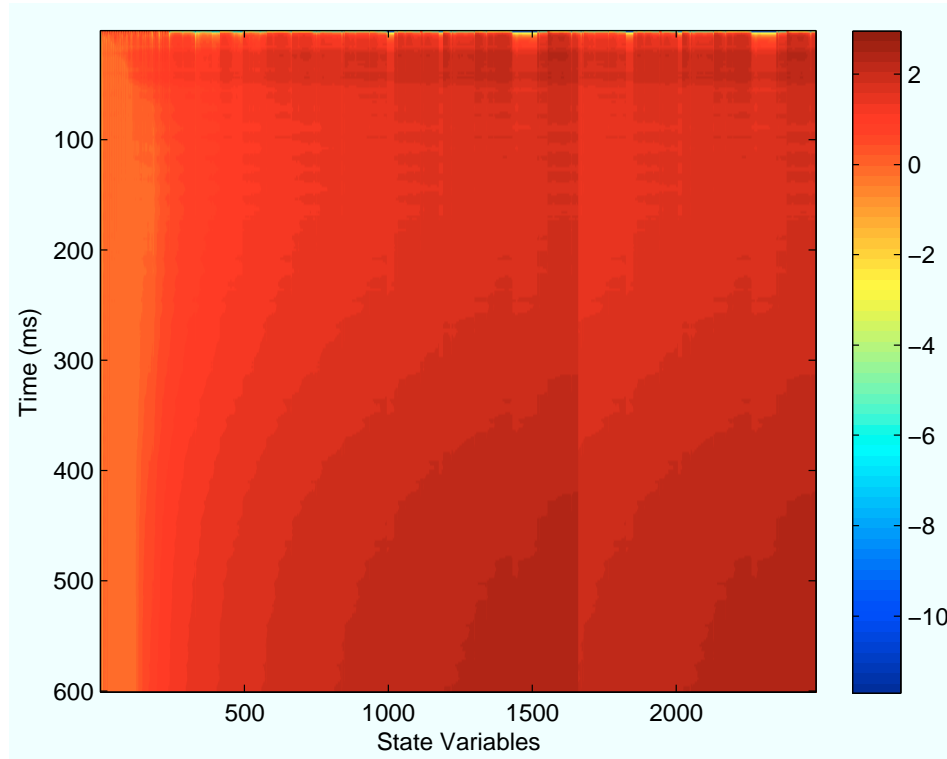

Figure 2: Time evolution of the Lyapunov exponents for the system in question.

4 Discussion

The code is written to be quite generic, and therefore the results presented here should be taken as an initial proof of concept. In the coming months I aim to study the effect that synaptic weights and delays have on the chaotic nature of the system. Specifically, in trying to address the original question which motivated this work, I'd like to compare networks with many weak connections to networks with fewer connections, albeit stronger connections.

Other areas to explore are the impact that delays have on the network. Clearly they increase computational time by exponentially increasing the size of the Jacobian matrix, but it has also been suggested that merely their presence in the system as represented mathematically by Equation 12 results in positive Lyapunov exponents. Perhaps a Laplacian transformation of the system can reduce the dimensionality of the system while preserving the delay aspect which is vitally important to a physiologically relevant representation of neural networks.

It is also important to consider that small isolated networks may be inherently chaotic, and that the predictability of their function can only be explained by chaotic coupling to other networks. This coupling could be mediated by activity carried in long range connections that has been observed to cause similar periodic activity is disparate brain regions with no known definitive purpose.

Acknowledgments

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