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BENG 525

4/22/19

Spiking Dynamics Observed in Three Neurons

ABSTRACT

A leaky integrate and fire (LIF) model is a basic mathematical model designed to simulate a neuron. The simplicity and relative ease with which network architectures can be constructed using patterns of LIF neurons make it a common choice for modeling. The RC circuit is used as the basis of the model. By way of algebraic manipulation, we can solve $I(t) = (u(t) - u(\text{rest})/R + C \cdot du/dt$, where $u - u(\text{rest})$ is the voltage across the resistor. The product of $C \cdot du/dt$ is the capacitive current. Through integration and Ohm's Law, we can derive membrane potential in a linear differential equation[1].

Delgado et al. aims to model the membrane potential of the LIF neuron through a random process known as Ornstein-Uhlenbeck. Through the random variable T_f , we can predict time between neuron firings. We can find an estimator of the average firing rate of the neuron[2].

The Nengo framework is an open-source neural modeling architecture leveraged across Python and the TensorFlow libraries, which are used for developing artificial intelligence models. Using LIF neurons, it is possible to create a high-level model of the brain[3]. More attention is spent by the modeler to conceptualize neural architecture; however, the drawback to

using the software is that we are unable to manipulate dynamics on the neuron-to-neuron scale, making Nengo more appropriate for large scale modeling.

METHODS

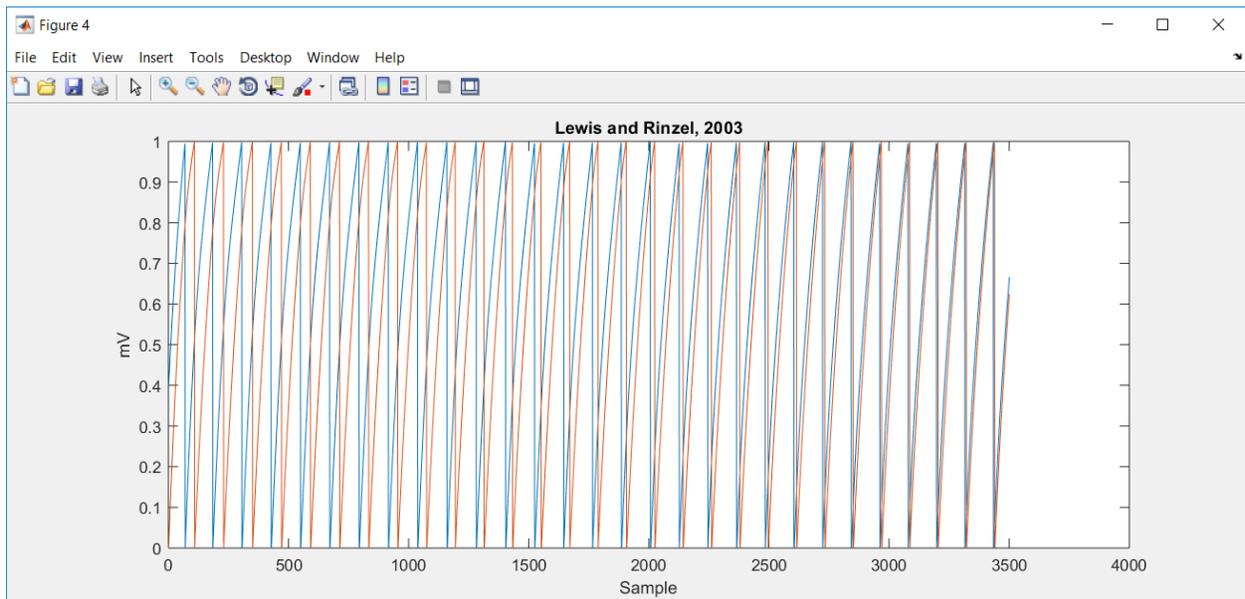
Lewis and Rinzel studied the dynamics of a pair of intrinsically oscillating LIF neurons[4]. We intend to use a MATLAB script first recreate the figures from Lewis and Rinzel's experiment to simulate different inhibitory and excitatory weights and their effect on the behavior of membrane potential over time. We then intend to observe the effects of this system. First, we validated the model by reproducing the figures from Lewis and Rinzer (2003).

We simulated two inhibitory neurons, each with an driving weight of -0.2. This created a matrix

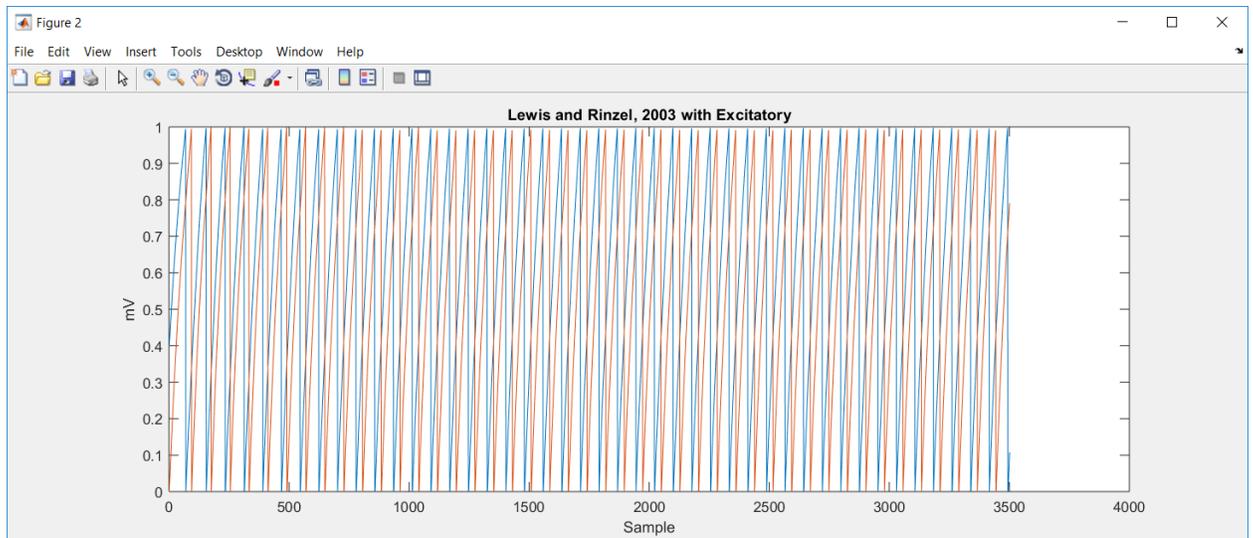
in the form: $\begin{pmatrix} 0 & -0.1 \\ -0.1 & 0 \end{pmatrix} * (x1 \ x2)$ where $x1$ and $x2$ are neurons. The coefficients describe the

behavior that each neuron has on the other. Applying an offset current of 1.1 mV to both neurons, while also applying an initial condition of 0.4 mV to $x1$ and 0 mV to $x2$, we produced the following graph. Over time, we see that the neuron potentials begin spiking closer together until

they appear to be simulating one another.

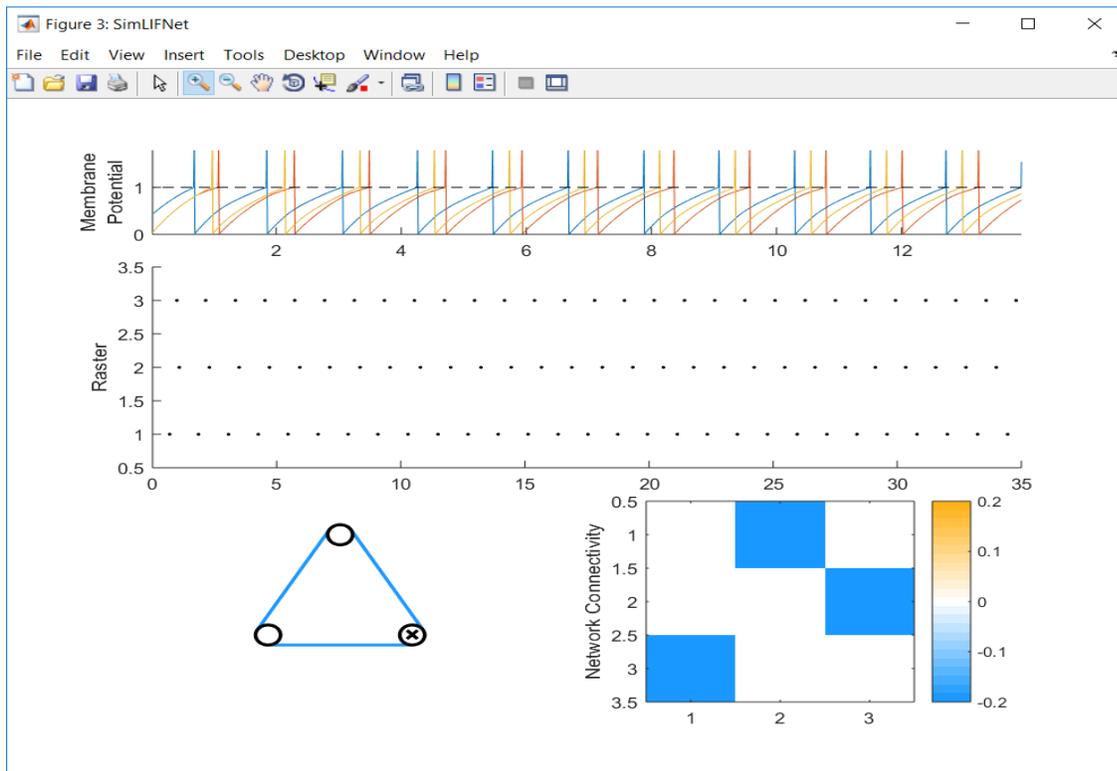


To investigate the effects of the driving matrix, we repeated the experiment, but reversed the signs of the matrix such that it took the form: $\begin{pmatrix} 0 & 0.2 \\ 0.2 & 0 \end{pmatrix} * (x1 \ x2)$. Applying all of the other default settings, we produced the following graph. Instead of slowing down one another until they were synchronous, we found that the neurons did not change their behavior throughout the course of the model.

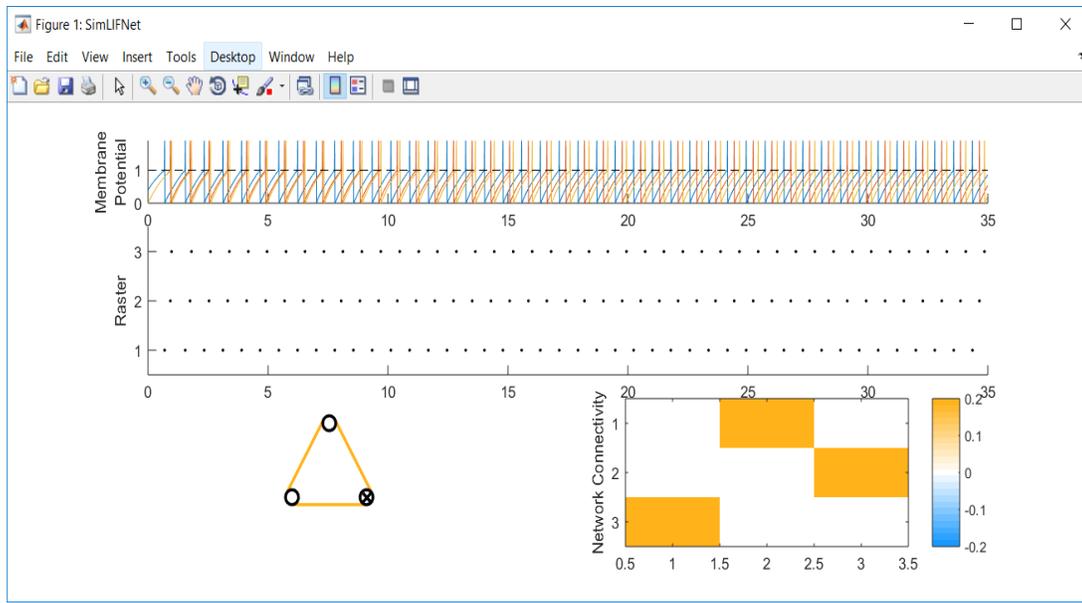


We then modeled a system of three neurons, each inhibiting one another. Neuron 1 inhibited Neuron 2 with a weight of -0.2 , Neuron 2 inhibited Neuron 3 with a weight of -0.2 , and Neuron 3 inhibited Neuron 1 with a weight of -0.2 . Neuron 1 had an initial condition of 0.4 mV, while Neuron 2 and 3 both had an initial condition of 0 . The following graphic shows the membrane potential, as well as the network connectivity matrix and a visual representation of the neurons affecting one another. We find that neuron 2 and 3 initially fire together; over time, however, the space between each neuron firing begins to increase. Eventually, all three are

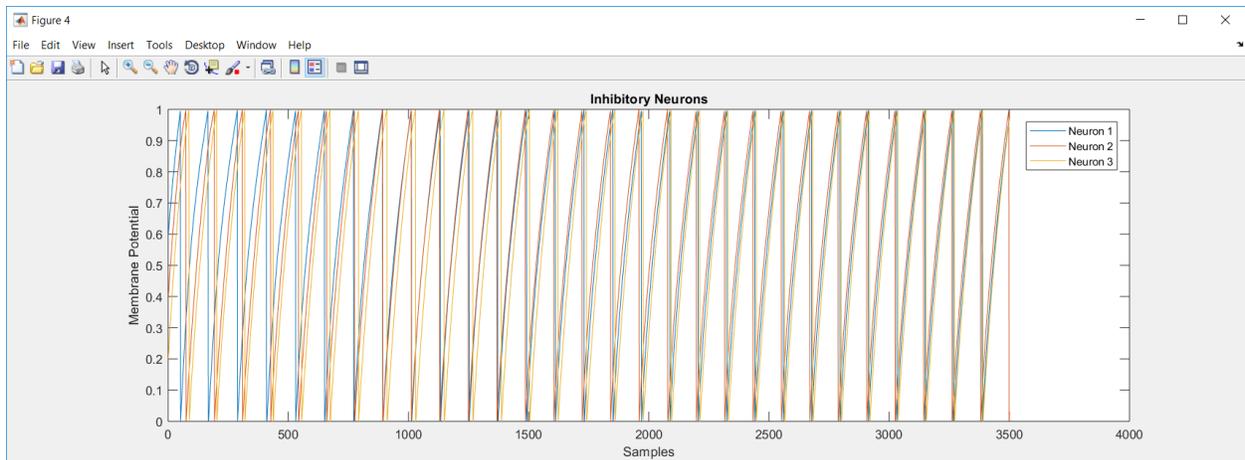
spiking at equidistant rates.



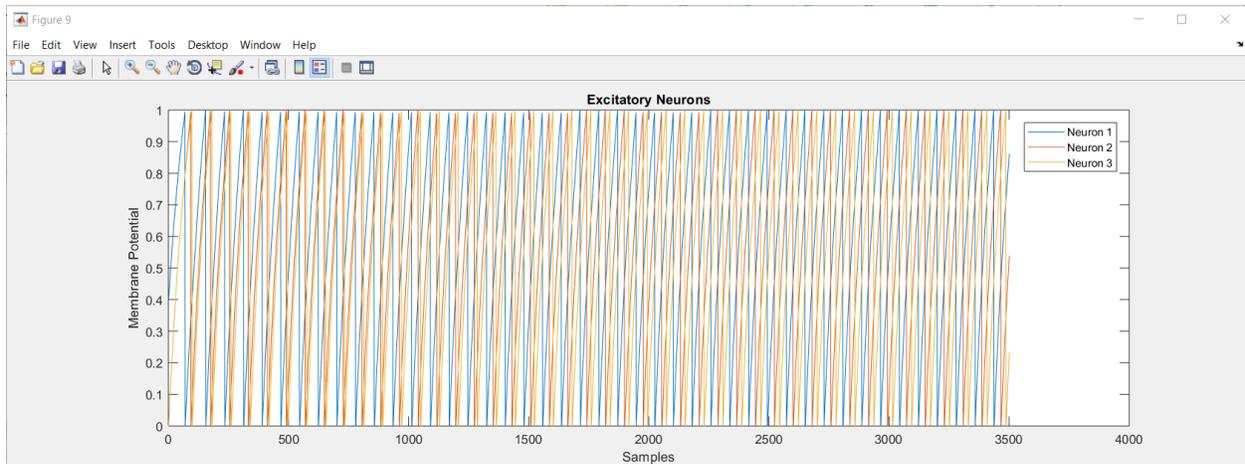
We repeated the same procedure we had in the last experiment, where the inhibitory/excitatory weight was inverted. The following graph was produced. It should be noted that the output is similar to the behavior of two excitatory neurons. In addition to a firing sequence that is 1.5x faster than its inhibited neurons, we observe membrane potentials that do not correct themselves. The raster plot included in both simulations gives us insight into the firing rate and sequence of both sets of inhibited and excitatory neurons.



Our last experiment involved testing the effect of inhibition and excitation on different initial conditions for three neurons. First, we replicated our original set up for the experiment, simulating three neurons that exhibit inhibitory behavior. We then changed the initial conditions of the first, second and third neuron to a membrane potential of 0.3, 0.5, and 0.6 mV at $t = 0$. Running the simulator, we found that the membrane potential over time followed the same behaviors as before. Over time, the time between membrane potential spikes became uniform. The figure below displays the behavior of the three neurons in greater detail. Note that around Samples = 2000, the membrane potential of all three neurons spike at identical distances.



Repeating the experiment with excitatory neurons, we kept the initial conditions the same. As we observed in our previous simulation, the raster plot indicated that the neurons fired at a multiplied rate of 1.5x. In addition to the accelerated firing rate, we observed a similar pattern in the membrane potentials over time, where the potentials from the different neurons did not correct themselves. The below figure displays the membrane voltage.



DISCUSSION

We successfully simulated a system of leaky integrate-and-fire neurons. With the addition of a third neuron, we were able to take steps towards understanding and generalizing inhibitory and excitatory dynamics across larger networks of LIF neurons. Our future work lies in studying behavior and dynamics across random network architectures using larger numbers of LIF neurons. Furthermore, a limitation of our current model is that we are not using a stochastic, random process to realistically simulate inhibition and excitation. Currently, our model uses a deterministic process for ease of simulation and representation. In addition, we are unable to study the effects of signal propagation through neurons with variable thicknesses of myelin sheaths; incorporating these physiological characteristics into our model is necessary so as to take steps towards engineering more realistic, while computationally inexpensive simulations of the neuron.

Works Cited

[1]

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[3]

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