Spiking Neurons

Computer Exercises using the NEURON Simulator

ANSWERS

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1. Frequency-Input Current (F-I) Firing Curve of a Neuron

Exercise 1.1: F-I curve of a simple neuron (FIcurve.hoc)

Question 1.1: What is the smallest current amp that causes the cell to fire repeated action potentials and how many does it fire in 100msecs?

Ans: *0.4 nA, 6 action potentials*

Question 1.2: What type (I or II) of F-I curve does this neuron have?

Ans: *Type II*

Exercise 1.2: Type I and Type II F-I Curves

Question 1.3: What cell parameters are different between the Type I and Type II examples?

Ans: *Type I cell has an A conductance and a higher leak reversal potential.*

Question 1.4 (supplementary question for later research): Can you explain why the difference in firing characteristics arises?

Ans: *The A-type potassium conductance in the Type I cell retards regeneration of the AP, leading to longer ISIs at just-threshold stimulation, so that firing rate increases slowly from 0 once threshold is reached. This is because it has a low activation threshold (so activates while the membrane potential approaches threshold) and it inactivates (so eventually it decreases, allowing the cell to fire an AP).*

2. Electrical Activity in a Hippocampal CA1 Pyramidal Cell Model

Question 2.1: What was the effect of K_A on the ability of a somatic AP to back-propagate into the dendrites?

Ans: *KA greatly reduces the amplitude of the BPAP with distance from the soma.*

Exercise 2.2: Synaptic input to SR

Example data:

Synapses:	10		50		100	
	Soma	SR	Soma	SR	Soma	SR
High K_A (0.03):	1.1mV	0.85	3.25	5.7	6	8.8
Low K_A (0.01):	1.35	1.35	5	7.9		

Question 2.2: Approximately, what is the relationship between the number of stimulated synapses and the EPSC amplitude?

Ans: *Very roughly linear.*

Question 2.3: Where in the cell is this action potential first generated?

Ans: *At the soma (we are not recording from the axon).*

Question 2.4: Now where in the cell is this action potential first generated?

Ans: *In the SR dendrites.*

Question 2.5: What is the cell's response now?

Ans: *Slow depolarisation in SR and soma, with second AP on top of this at the soma.*

Question 2.6: What characteristics of the VGCCs can you deduce from these simulations?

Ans: *They contribute to the slow depolarisation, so they are activated by depolarisation and generate a slower current than the Na channels that underpin the AP.*

Question 2.7: How do these EPSC amplitudes compare with when the K_A density was high?

Ans: *The amplitudes are all higher.*

Question 2.8: What happens now? Design and run a simulation to test your conclusion.

Ans: *Single AP followed by a slow and large depolarisation, likely due to full VGCC activation ie a calcium spike. Reducing the Ca-R conductance to 0 confirms this.*

Exercise 2.3 (supplementary): Synaptic input to SLM

Question 2.9: What are your conclusions?

Ans: *In low KA, 120 activated synapses in SLM will generate a somatic AP. In high KA, even 500 active SLM synapses cause only a small voltage deflection in SR and the soma (note that 500 is the maximum number of synapses possible without editing run_PC.hoc).*

3. Simple Excitation-Inhibition (E-I) Oscillator

Question 3.1: What different firing patterns emerge?

Ans: *0.6: No spiking; 0.7: spike doublets at low freq; 0.8: longer bursts; 0.9: high freq train*

Question 3.2: Again, what different firing patterns emerge?

Ans: *0.01, 0.06: single spikes; 0.02-0.05: rhythmic bursting*

Question 3.3: What happens now to the firing patterns?

Ans: *0.004: single spikes; 0.005-0.007: bursting; 0.008: low freq single spike*

Question 3.4: Again, what happens to the firing patterns?

Ans: *0: single spikes; 1-3: bursts; 4: single spikes*

Question 3.5 (summary of results): What is the general behaviour of this circuit and how do the various parameters (weights, delays etc) affect this behaviour?

Ans: *Rhythmic burst firing emerges within a narrow range of each parameter. The alternatives are either high frequency regular spikes (equivalent to unconnected net) or low frequency regular spikes (most prominent at high I->E weights).*

4. Excitation-Inhibition Balance

Exercise 4.1: E-I balance in a single I&F neuron

Question 4.1: How sensitive is irregular firing to the balance between excitation and inhibition?

Ans: *Quite sensitive.*

Increasing JE produces more regular high freq firing (eg 1.5 still irregular; 2.0 largely regular). Decreasing JE (0.09) produces lower frequency irregular firing (note firing stops if JE=0.08, with JI=-0.2).

Small increases in JI amp (to -0.23) produces low frequency irregular firing. Firing stops for larger JI amp. Firing becomes increasingly regular with decreases in JI amp eg to -0.1. Reducing JE to eg 0.07 with JI=-0.1 restores irregular firing.

Sufficient inhibition for any level of excitation will give irregular firing.

Exercise 4.2: E-I Balance in a Network of I&F Neurons

Question 4.2: How do the network firing characteristics change as the size of the network is reduced?

Ans: *A net of size 0.1 exhibits long periods of high frequency regular firing, interspersed with short periods of low frequency irregular firing.*

5. Spike-Time-Dependent Plasticity (STDP) in Action

Exercise 5.1: Phase precession of spike timing

Question 5.1: Do you see any change in the timing of the postsynaptic spike relative to the inputs over the course of the simulation?

Ans: *The spike time gets earlier with respect to the inputs.*

Question 5.2: What is the pattern of weights across the inputs 0 to 9?

Ans: *The first 3 weights are relatively large (have been potentiated as inputs are consistently just before the output spike). Later weights remain small as these inputs quickly end up occurring later than the output spike.*

Question 5.3: What differences can you see in the voltage trace and the weight distribution from previously?

Ans: *Similar precession of output spike time; more sharp distinction between first 3 potentiated weights and others (that remain even smaller).*

Question 5.4 (summary): How and why does the timing of the postsynaptic spike change, and why does a pattern emerge of weights across the different inputs?

Ans: *Timing of input spike relative to the output spike determines whether the input weight is potentiated or depressed. Rate of output spike precession depends on magnitude of LTP / LTD.*

Exercise 5.2: Sequence learning

Question 5.5: Can you see a pattern in the weights that have developed across the neurons?

Ans: *Each neuron connects strongly to the next neuron (in order), with progressively weaker connections to others (in order 0, 1, 2 etc).*

Question 5.6: Can you see any difference in the pattern of weights now?

Ans: *Now the weights to preceding neurons are also depressed.*

Question 5.7: How do you think this delay might interact with the time windows of LTP and LTD (see the STDP figure above)?

Ans: *Delay will affect the magnitude of weight changes, according to the STDP curve.*

Question 5.8 (summary): What patterns of weights emerge? Can you explain why? And how do the patterns vary with different rates of LTP and LTD and with different delays between spikes?

Ans: *Summary of above.*

6. Associative Memory in a Network of Spiking Neurons

Question 6.1: Which of these 3 cells do you think belong to the stored pattern and which do not?

Ans: *Cells 7 and 30 (top 2 plots) do belong to the pattern; cell 0 (bottom) does not.*

Question 6.2: How sensitive does pattern recall seem to be to these network parameters?

Ans: *Overall, it is reasonably sensitive.*