Model Neuron Summary

The Connor-Stevens model neuron is similar to the Hodgkin-Huxley model in that they share almost all the same conductances and variables; the difference is the addition of another potassium current called the A-current which has its own gating variables and channel. The neuron model consists of a leak channel, Na channels, K-channels, and A-channels. The leak channel is a variable that represents all other types of ion movement that isn't occurring through the voltage-gated channels. It's always open, allowing potassium ions to leak out passively. The leak reversal potential contributes to the neuron's resting membrane potential. Below is a simulation of a firing neuron with an applied current of 100 picoamps over the course of 100 milliseconds. What's unique about this neuron is that the course the action potential takes is more fluid; the only major oscillation that's occurring is the main spike of the action potential. This is a Type-1 neuron, which does not have a rebound spike or discontinuity in the transition between the resting phase and depolarization.

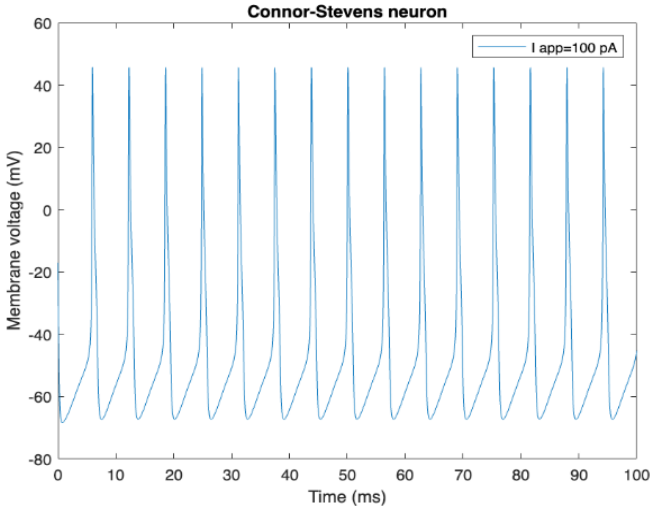

Figure 1: A firing neuron after a current of 100 pA is applied.

The neuron's sodium channels open in response to a stimulus that is able to pass the threshold voltage. The channels are voltage dependent; a higher voltage means more open sodium channels. The proportion of open channels at any given moment is the conductance. The maximum sodium conductance is 12000 microsiemens, which is a high conductance relative to the other ions' conductances. The channel's activation is represented by the m-gate and deactivation is represented by the h-gate. Alpha is a rate constant that determines the speed with which a channel opens, while beta is a rate constant that determines the speed of the channel's closing. Both the m and h gates have their own specific rate constants which change depending on voltage. Sodium channels are quick to open and close. The sharp influx and halt of inward sodium current shapes the dynamic nature of the action potential. At the opening of the sodium gates, sodium rushes into the cell and depolarizes it. The cell's membrane voltage increases and peaks at about 55 mV, the value of sodium's reversal potential. Then, there is a short window of time called the absolute refractory period that occurs after the peak of the action potential. This is when action potentials cannot be made because the sodium channels are deactivated. Sodium channels eventually reactivate with time.

The K-channel is one type of potassium channel that this model neuron has. Its maximum conductance is 2000 microsiemens and its reversal potential is -72 mV. The neuron is the most

conductive to the K-current right after the peak of the action potential. After sodium finishes rushing into the cell, K-channels open to let potassium out. This hyperpolarizes the cell. K-channel activation is represented by the n-gate. Like sodium's gates, the n-gate has rate constants as well. The n-gate is slow to close; this delay causes the undershoot phase of the action potential.

The A-type channel is the second potassium channel my neuron has. It has a conductance of 4770 microsiemens and a reversal potential of -75 mV. It activates and deactivates like the sodium channel. The a-gate activates the flow of A-current and the b-gate deactivates. Like the other gates, a and b are affected by the value of tau, its rate constant. The A-current is like the K-current because they are potassium currents that flow out of the cell after the peak of the action potential, causing the cell to hyperpolarize. What makes the A-current unique is that it activates at lower membrane potentials and deactivates at high membrane potentials. This contributes to the neuron's ability to produce action potentials at low firing rates, in addition to its parameters. The default parameters as defined by Connor and Stevens make the neuron more excitable than the Hodgkin and Huxley neuron. Below is a figure representing the gating variables, a and b, when the neuron is at rest. A neuron with a higher resting membrane potential will have more activated a-gates, therefore it will have a high A-current conductance and decreased excitability. A neuron with a lower resting membrane potential will still have A-current conductance, but not as high. Regardless, conductance is non-zero even when the resting membrane potential is as negative as -100 mV, meaning the A-current is still able to have an inhibitory effect on the membrane potential.

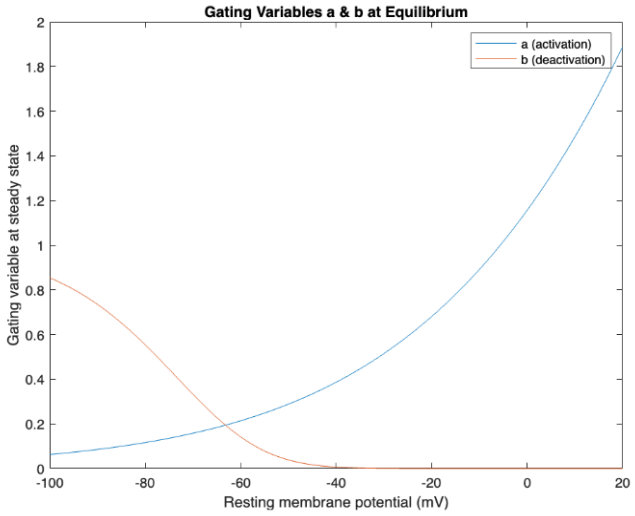

Figure 2: Activation and deactivation mechanisms of the A-current at different resting membrane potentials

The representation of deactivation (as opposed to the K-current only having an n-gate for activation) makes it possible to establish a set window of activation, where ion movement is limited to that window period only. a and b are voltage dependent, meaning that A-type current activity depends on the membrane voltage of the cell. The range of voltages where the A-type current is active is slightly lower than that of the Na channel. This translates to the A-current being slightly activated after the peak of the action potential. Since the A-current is hyperpolarizing, the additional negative charge suppresses rebound spikes and oscillations in the membrane voltage. (Miller, 2018)

Source: Miller, P. (2018). *An Introductory Course in Computational Neuroscience*. MIT Press.