COMPLEX SINGLE CELL MODELS

Adaptation

- 'Regular spiking' cells most common in cortex
- Adaptation occurs because of a slow hyperpolarizing K current in these cells



Adapting LIF circuit

• A variable resistor, R_{adapt}, can account for this K



aLIF Behaviour

- Adapt channels stay closed until a spike, then they open (by decreasing R_{adapt}) essentially lowering the reset voltage, making it harder for the next spike to be fired.
- Between spikes, the channels start to close, raising R_{adapt} at a speed determined by τ_{adapt}.

$$\begin{aligned} \frac{dV}{dt} &= -\frac{1}{\tau^{RC}} \left(V \left(1 + \frac{R}{R_{adapt}} \right) - J_M R \right) \\ \frac{dR_{adapt}}{dt} &= \frac{R_{adapt}}{\tau_{adapt}} \end{aligned}$$

• Comparison of aLIF response function to a complex conductance model



aLIF decoding

• aLIF (as we will see) is more efficient



Ion flow: rest





Inside the Neuron



B: Na open C: K open D: Na close E: Na pump F: K close G: Pumps

K-Na pump

http://tx.technion.ac.il/~yarmola/bioPhys/Nerve%20impulse%20propagation.htm

- Absolute: Na channels are open or recovering. No second spike
- Relative: K channels are open memberane is hyperpolarized (-80mV), so hard to generate 2nd spike (usually from -70mV)



Hodgkin-Huxley circuit



Inside Membrane

Class II circuit

Hodgkin-Huxley equations

• 4D nonlinear differential equation

$$C\frac{dV}{dt} = -g_{Na}m^{3}h(V - E_{Na}) - g_{K}n^{4}(V - E_{K}) - g(V - E) + J_{M}$$

$$\frac{dm}{dt} = \frac{1}{\tau_{m}(V)}(-m + M(V))$$

$$\frac{dh}{dt} = \frac{1}{\tau_{h}(V)}(-h + H(V))$$

$$\frac{dn}{dt} = \frac{1}{\tau_{n}(V)}(-n + N(V)).$$

HH parameter dynamics

• *m*, *n* activation; *h*, inactiviation params

all approach some asymptote (e.g., N(V)) with a time constant (e.g., τ_n).



HH Dynamics: Hopf



Class 1 circuit

• zero minimum spiking hz and grow monotonically

need to add A-current, another fast K current



Inside Membrane

Theta neuron behaviour

- Canonical model of saddle-node bifurcation
- Phase variable maps to neural states



Theta neuron decoding

• Takes 100x longer than LIF to run



Wilson Neuron (reduced)

• Start with a HH neuron model. Rinzel simps:

- Na activation is very fast (\tau(V)) is really small), so allow m=M(V) (no dynamics)
- Na inactivation is equal and opposite to K activation, so let h=1-n (combine h & n -> R)
- We need the A-current. Rose & Hindmarsh simp:
 - Make the dynamics for R quadratic.
- Include adaptation variable with slow dynamics
- Have 3D class I model!

Wilson neuron equations

$$C\frac{dV}{dt} = -\left(1781 + 4758V + 3380V^2\right)(V - 48)$$

$$-26R(V + 95) - 13H(V + 95) + J_M$$

$$\frac{dR}{dt} = \frac{1}{5.6}\left(-R + 129V + 79 + 330(V + 38)^2\right)$$

$$\frac{dH}{dt} = \frac{1}{99.0}(-H + 11(V + 75.4)(V + 69)).$$

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• Comparison of Wilson neuron and real data



 Pros: it's a class I, adapting neuron with spike dynamics; captures spike height changes, spike shape, and after-hyperpolarizations (overshoot of the resting value after a spike)

• Cons: 600x slower than LIF

Wilson neuron decoding



Comparíson of neurons

Neuron	Rate	Bits/spike	RMSE	Run time (s)
LIF	114	1.24	0.153	0.18
Adapting LIF	114	2.23	0.153	0.24
θ -Neuron	109	0.96	0.160	20.1
Wilson Model	91	2.00	0.186	125.2

Summary

- variety: `phenomenological' models through to more complete models that include adaptation, spike dynamics, and ion channel dynamics.
- We haven't discussed are compartmental models. (<u>http://diwww.epfl.ch/~gerstner/SPNM/node17.html</u>).
- All of the models have info rates between 1-3 b/s
- Adaptation seems to help improve efficiency (using Gaussian white noise here)
- LIF are very computationally efficient and have reasonable info trans efficiency.