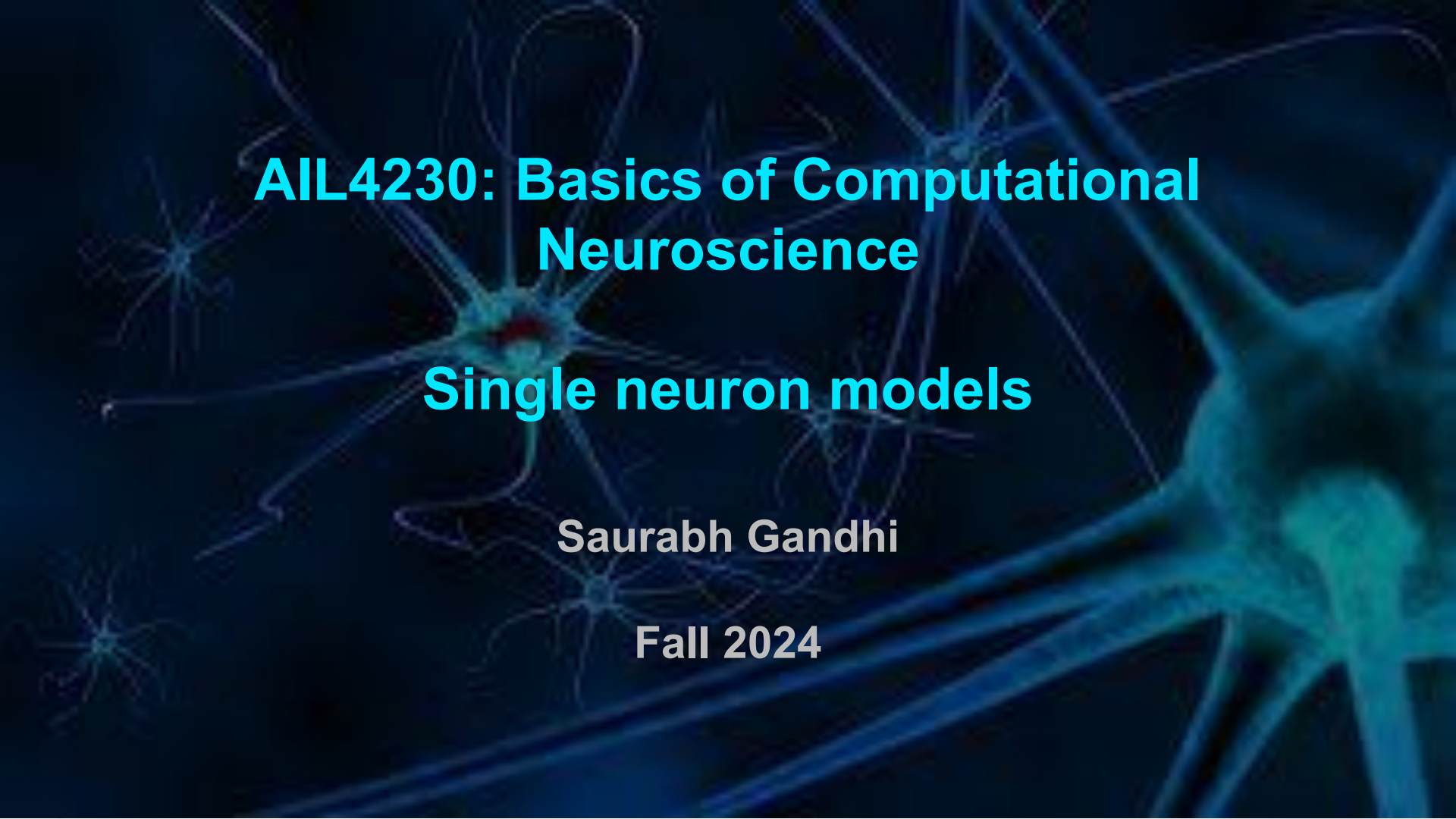


AIL4230: Basics of Computational Neuroscience

Single neuron models

Saurabh Gandhi

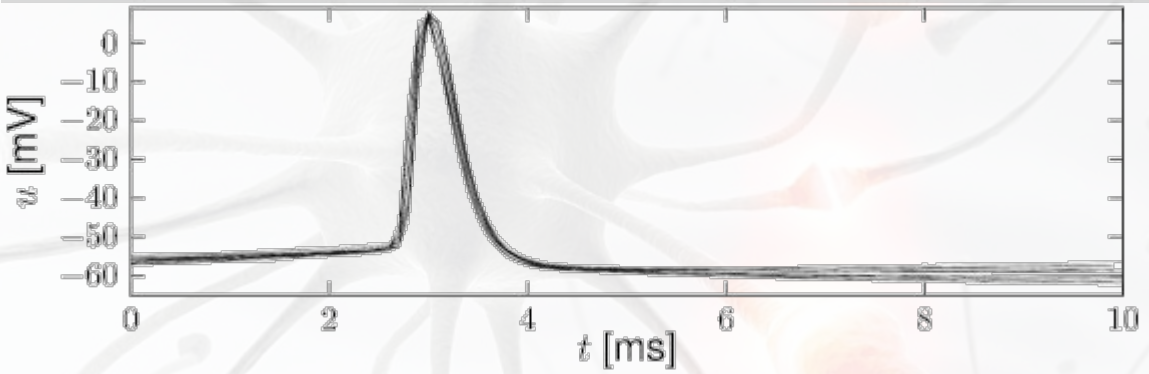
Fall 2024

Overview of topics

Phenomenology of a neuron

What peculiarities of a neuron do we care about to model?

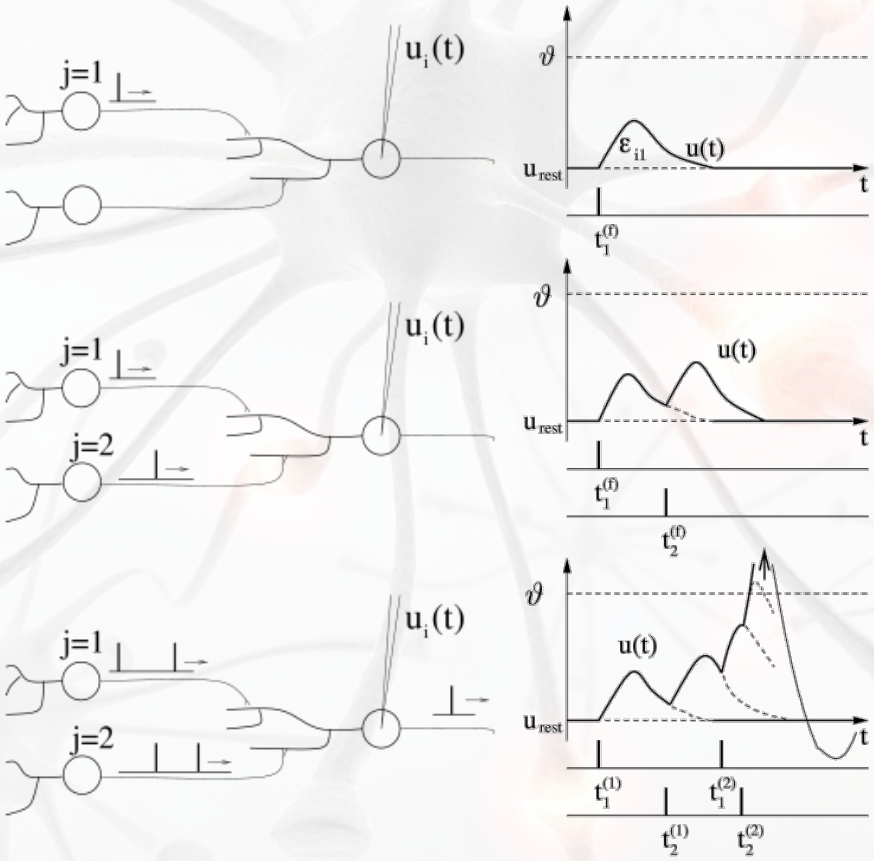
The action potential



~100 mV, 1-2 ms in duration

- Action potentials are the elementary unit of information transmission
- Shape does not change as it propagates
- Typically series of APs are fired by a cell
- Information not contained in the shape, but in the timing / rate of APs
- Refractoriness: APs are well-separated; when one AP has just fired, a second AP cannot fire immediately for some time

Integration of signals at synapses



- Excitatory and inhibitory postsynaptic potentials ~1mV
- PSPs add linearly for few simultaneous inputs
- Linearity breaks down when too many spikes arrive at a time (~20-23 mV)
- Sudden spike followed by a hyperpolarization called ‘spike-afterpotential’
- Typically membrane potential fluctuates below threshold; rarely reaches threshold causing a spike!

Integrate and fire model – a phenomenological model

What is a phenomenological model?

A model that replicates a phenomenon without caring for the mechanism.

Can you summarize the phenomenon we are trying to model? In other words, which of these observations should our model try to capture?

1. Linear summation of input upto a certain threshold
2. Non-linear dependence of output on input beyond the threshold
3. Duration of the action potential
4. Shape of the action potential
5. Rate of firing of action potentials for a given input
6. Refractoriness

Integrate the input, and fire if it crosses a threshold

Integrate and fire model

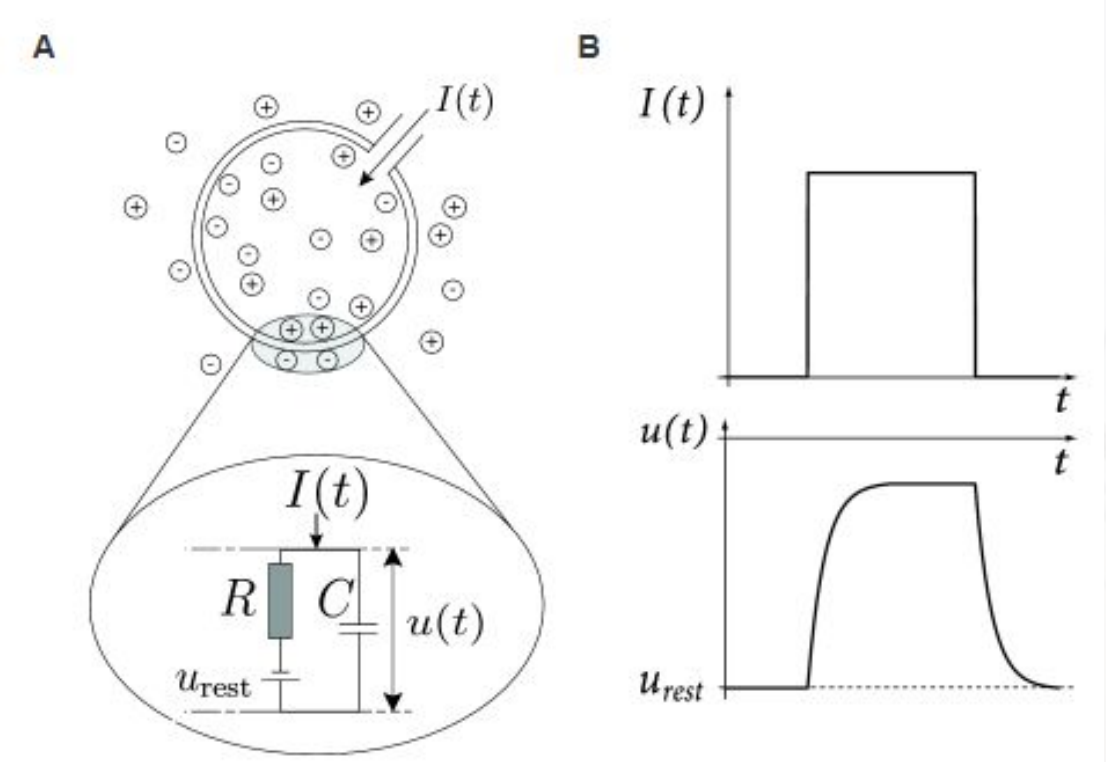
Integrate-and-fire models have two separate components that are both necessary to define their dynamics:

1. An equation that describes the evolution of the membrane potential ui(t)
2. A mechanism to generate spikes

The Leaky Integrate and Fire model:

1. A linear differential equation to describe the evolution of the membrane potential
2. A threshold for spike firing

Leaky Integrate and Fire model



- Cell membrane is a capacitor
- Ions can flow through channels, acting like a resistor
- Ion pumps maintain a potential difference across the membrane, like a battery
- Classic RC circuit model
- External current charges the capacitor, and it gets discharged through the resistor
- Fixed voltage threshold: for spiking
- Voltage is reset after spike

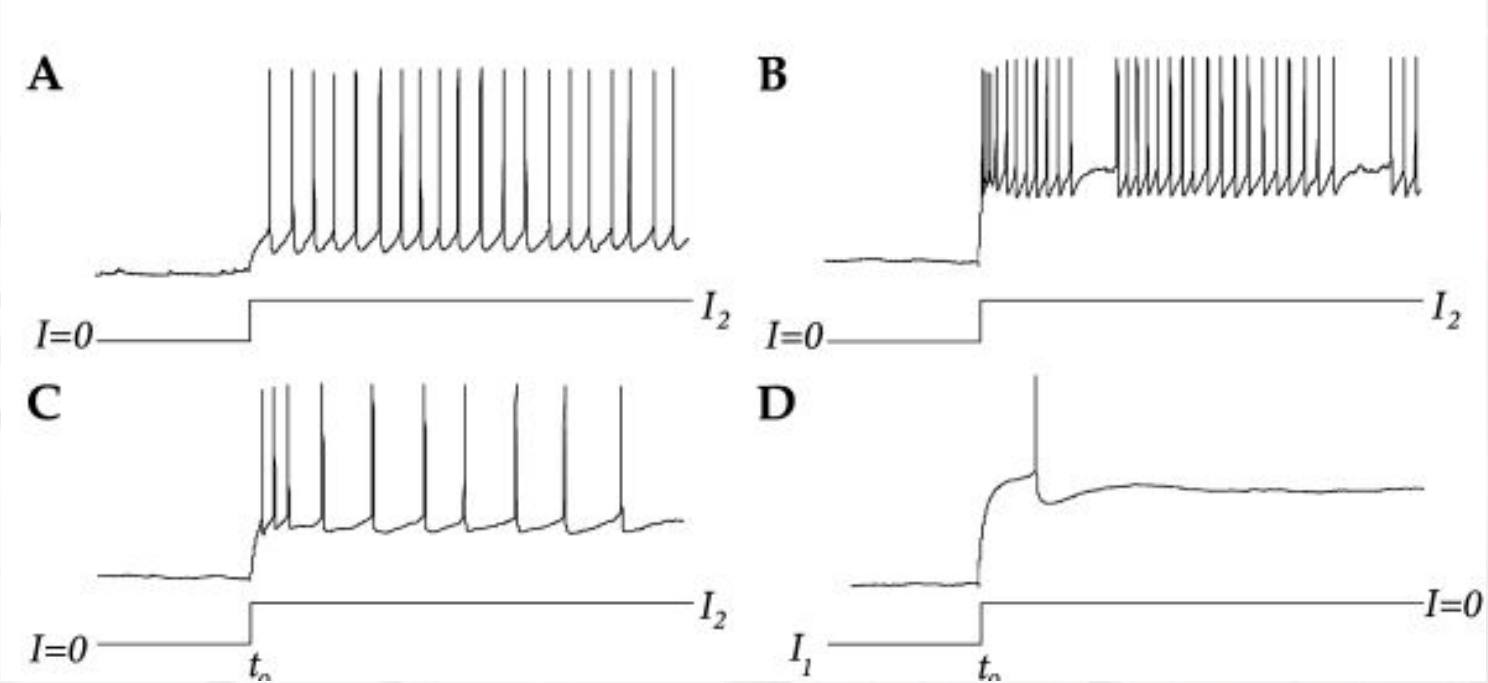
Limitations of LIF model

- Membrane potential is reset, removing all history of the previous spikes!
- Inputs are always added linearly

Let us look at more phenomenology

- Interaction between spikes – arrival of a spike can change the impact of the next spike input
- Adaptation – Generation of an output spike can change the subsequent generation of output spikes

More phenomenology of neurons – Adaptation



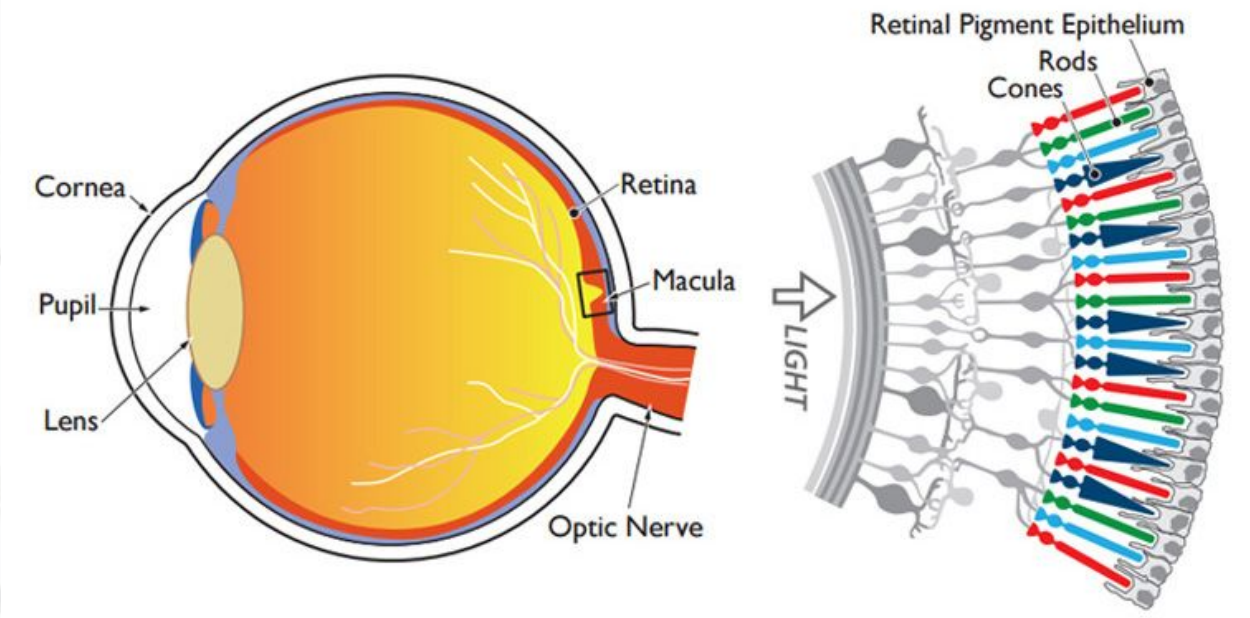
Regular spiking neurons: show adaptation (C)

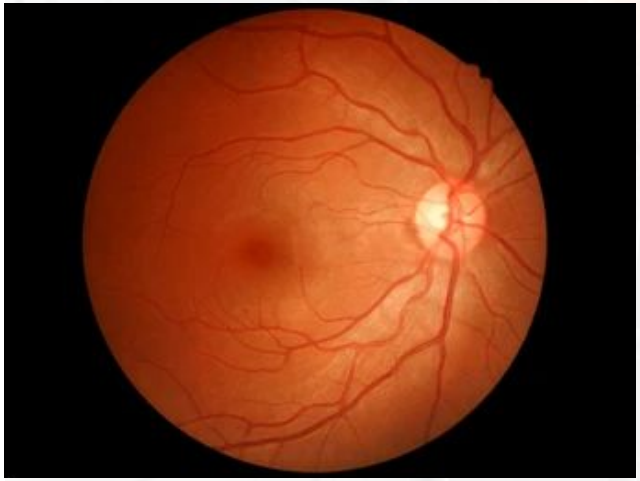
Fast spiking neurons: no adaptation (A)

Bursting and stuttering neurons (B)

Inhibitory rebound (D)

Let’s do some experiments, right here!



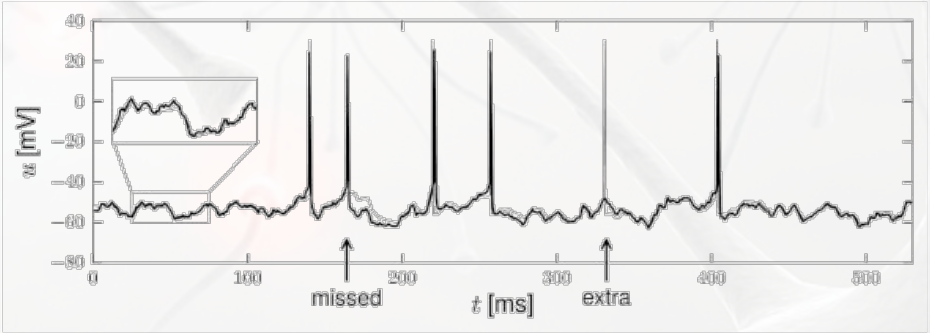


- Why are the light sensitive cells not on the surface but deep under?
- Why don’t we see the shadow of the blood vessels?
- Can you think of an experiment RIGHT NOW to see the blood vessels in your own eye?

How can we get adaptation?

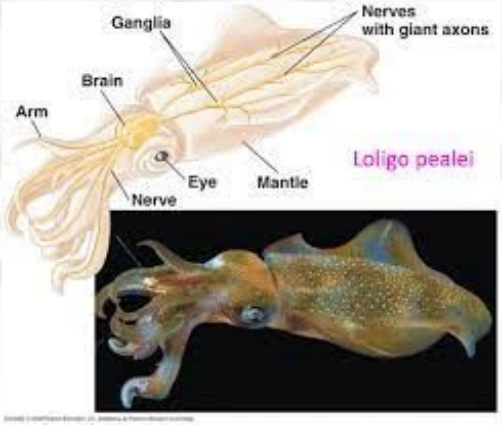
Go to the mechanisms!

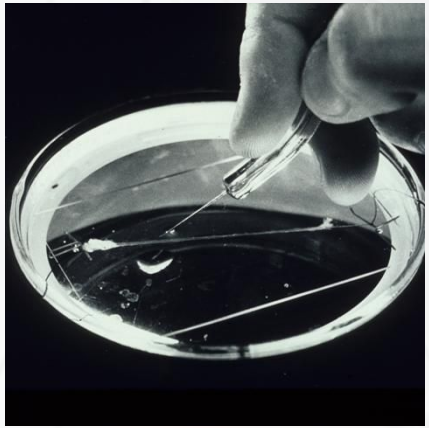
- A ‘fast’ timescale that generates spikes based on the ‘state’ of the neuron
- A ‘slow’ timescale that changes the state of the neuron over a longer period of time
- Biophysical models can naturally incorporate these different timescales – next topic, Hodgkin Huxley model
- Can incorporate two timescales in a phenomenological model also – make the spiking threshold dynamic

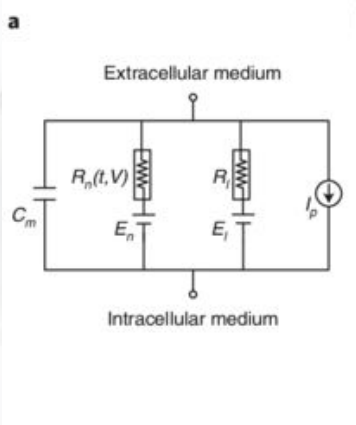


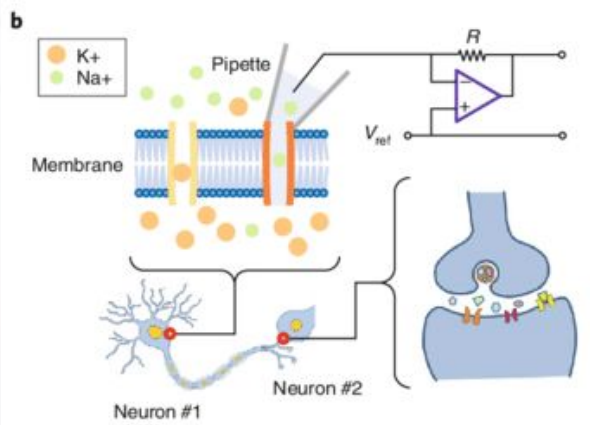
Hodgkin-Huxley Neurons

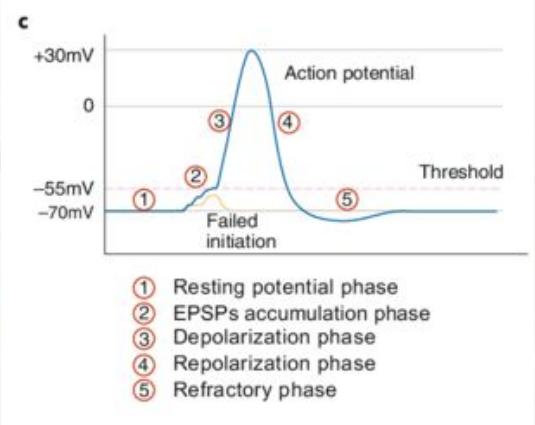
Currents through ion channels cause action potentials











It is all about the ions…

The cell membrane has different densities of ions on both sides. We need to understand them!

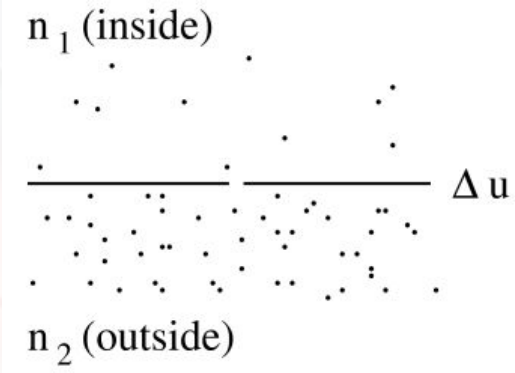
Consider a membrane with a hole, and ions on both sides, with a potential difference across the membrane

What direction will the ions flow?

Can we tell what the ratio of ions inside and outside is?

Probability of a molecule to take a state of energy E is proportional to the Boltzmann factor p(E) ∝ exp(−E/kT)

Nernst potential, also called reversal potential



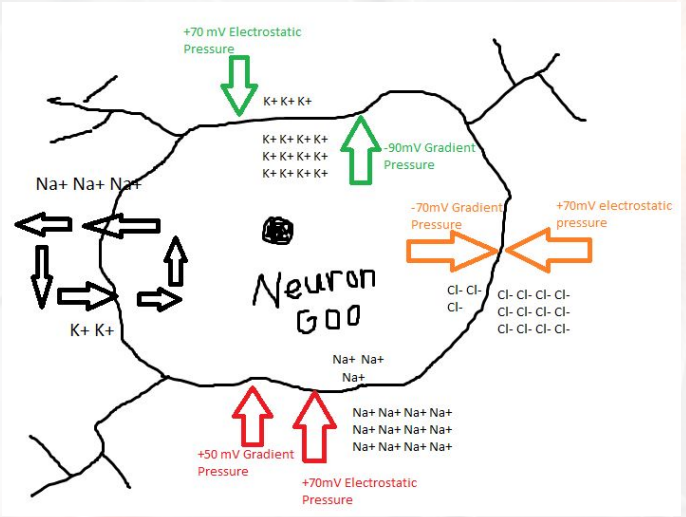
$$\frac{n(x_1)}{n(x_2)} = \exp\left[-\frac{q\,u(x_1) - q\,u(x_2)}{kT}\right]$$

$$\Delta u = \frac{kT}{q} \ln \frac{n_2}{n_1}$$

Understanding ion flow

$$\Delta u = \frac{kT}{q} \ln \frac{n_2}{n_1}$$

Ion	Intracellular concentration	Extracellular concentration	Nernst potential
Na^+	10 mM	145 mM	+67 mV
K^+	140 mM	5 mM	−83mV



At resting state, the membrane potential is $u_{rest} \approx$ -65 mV

What direction do the ions tend to flow?

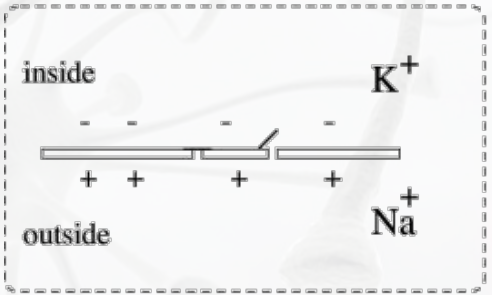
Are Na and K ions at equilibrium?

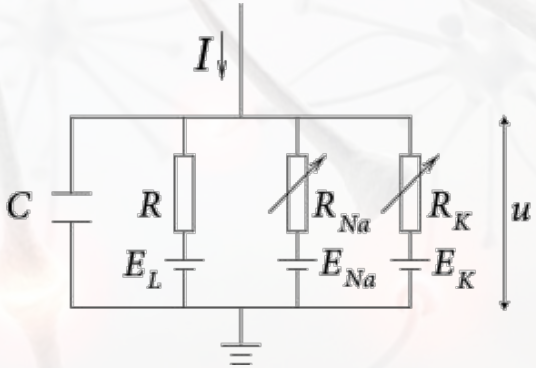
What stops them from coming to equilibrium?

What happens when you increase the temperature?

The Hodgkin Huxley experiment

- Hodgkin and Huxley performed experiments on the giant axon of the squid and found three different types of ion current:
  - sodium, potassium, and a leak current that consists mainly of Cl− ions
- Specific voltage-dependent ion channels, one for sodium and another one for potassium, control the flow of those ions through the cell membrane
- The leak current takes care of other channel types which are not described explicitly
- HH measured how the effective resistance of a channel changes as a function of time and voltage





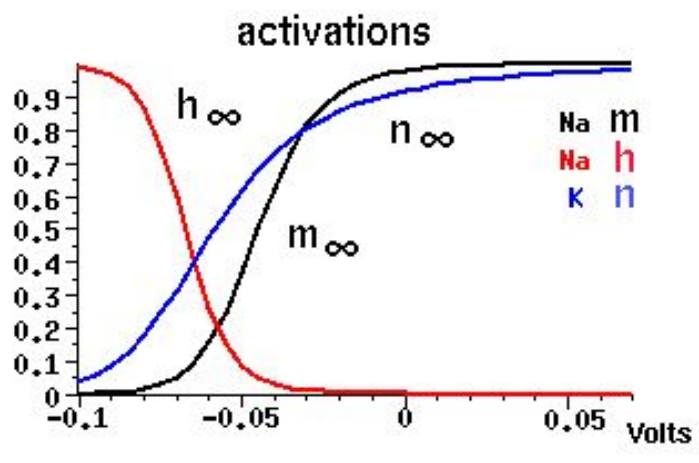
- What are the 3 different batteries?
  - Nernst potential for different ions
- What are the arrows across resistors?
  - Resistances are not constant (depend on opening and closing of channels)

The Hodgkin Huxley Model

$$\sum_k I_k = g_{\mathrm{Na}}\, m^3 h\, (u - E_{\mathrm{Na}}) + g_{\mathrm{K}}\, n^4\, (u - E_{\mathrm{K}}) + g_L\, (u - E_L)$$

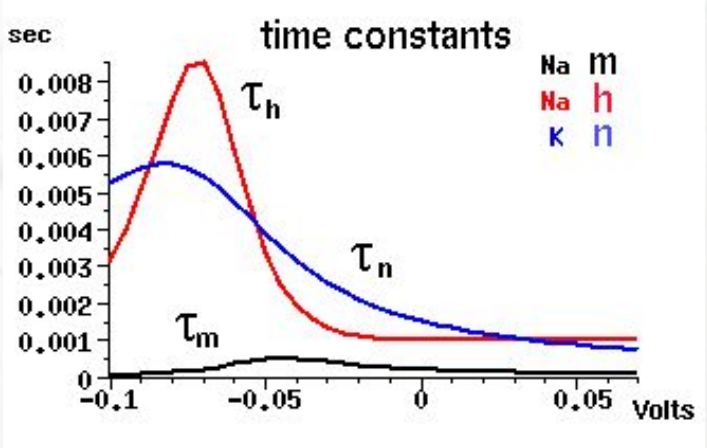
x	E_x [mV]	g_x [mS / cm^2]
Na	55	40
K	-77	35
L	-65	0.3

m*, *h* and *n* are not constant, but depend on *u



AND: they do not change instantaneously, but slowly!

$$\dot{x} = -\frac{1}{\tau_x\,(u)}\,[x - x_0\,(u)]$$

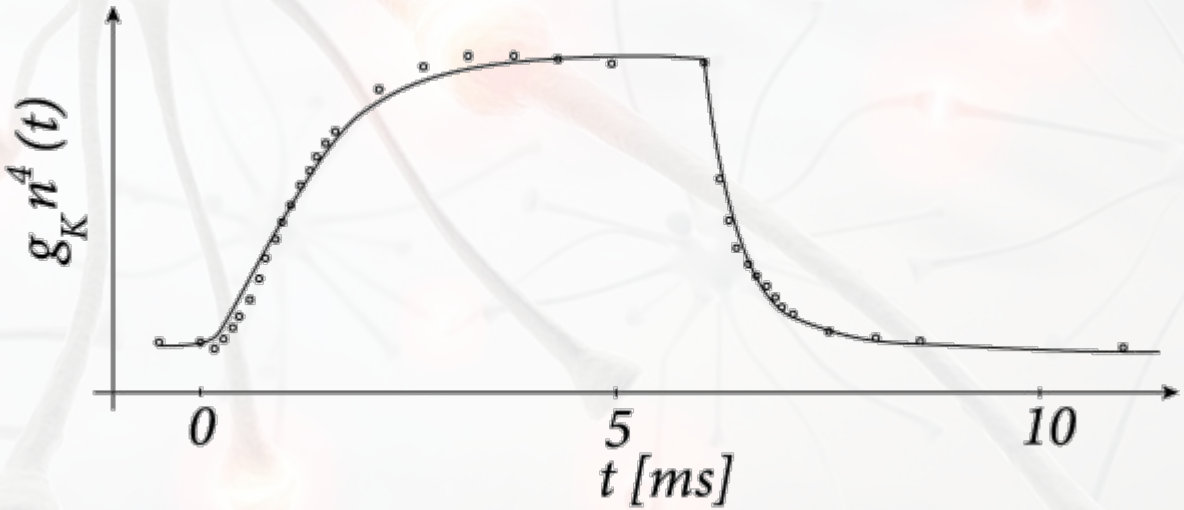


How can we measure *x(u)* and *τ(u)*?

Measuring HH parameters

Assume the cell is held at some constant membrane potential.

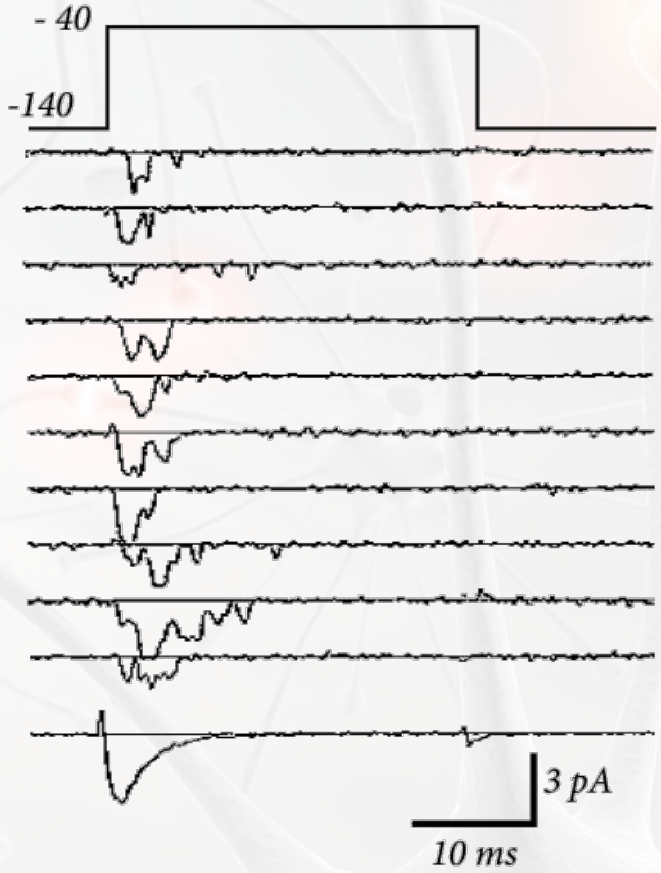
Now apply a constant current and allow it to come to a different constant potential.



Interesting phenomena in the HH model

- Spiking threshold?
  - Is there a voltage threshold?
  - Charge threshold?
  - Current threshold?
  - f-I curves
- How does one spike affect consequent neuron behavior?
  - What causes refractoriness?
    - Hyperpolarizing spike after-potential
    - Resistance of the membrane is reduced compared to the situation at rest
  - How would you characterize refractoriness?
- Rebound spiking
- Stochastic channel opening

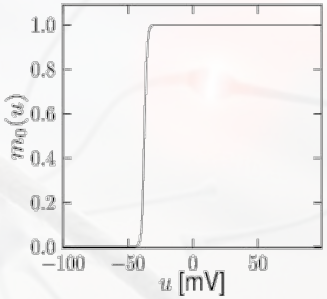
Explore these in the simulation assignment!

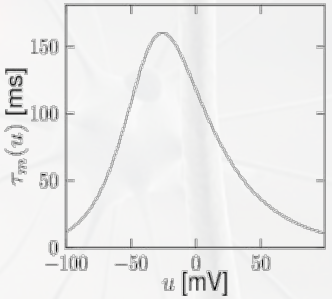


How to get adaptation?

Need two timescales – fast timescale causes spiking, and slow timescale changes the physiological parameters that determine spiking ‘threshold’

- Refractoriness is caused by the slow return of the sodium inactivation and the potassium activation to their equilibrium values
- Typical time scale of recovery in the Hodgkin-Huxley model is 4 ms for the potassium channel activation and 20 ms for the sodium channel inactivation
- What if either of these happened much much slower?
- Adaptation as prolonged refractoriness – the I_m current

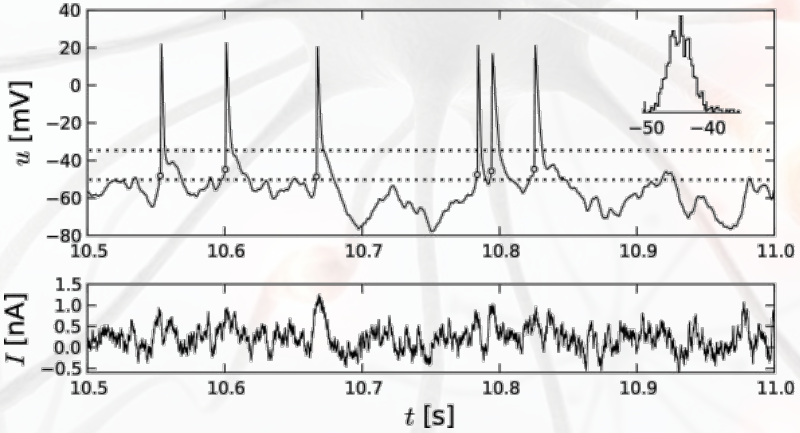


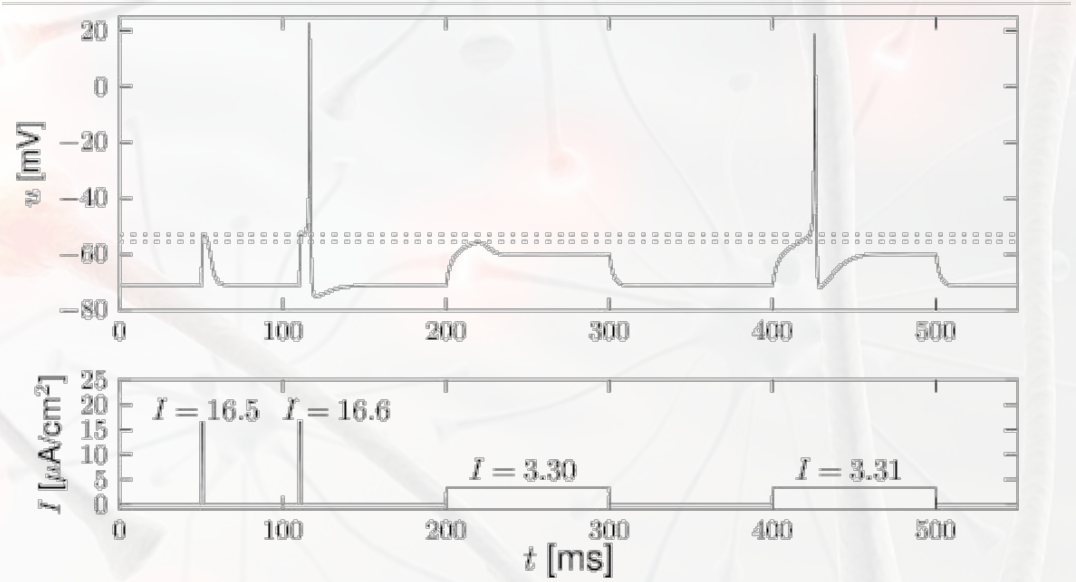


- Adaptation caused by slow calcium influx
  - Spike → Influx of Ca^{2+} (fixed amount per spike)
  - Calcium dependent potassium channels → open when [Ca^{2+}] increases above a threshold
  - Calcium ions diffuse out of the cell (calcium extrusion)

$$n_0 = \frac{k_2 e^{k_1[Ca]}}{1 + k_2 e^{k_1[Ca]}}$$

Threshold effects in neurons





A note on assignments

What is the purpose?

A. Evaluation – It is like a quiz!

B. An opportunity to apply what we learnt in class and deepen our understanding

C. Copy and get some free marks

D. A means for instructors to take out their grudge on students

Why you should attempt it honestly

- Moral / ethics: Being honest in our efforts is what distinguishes us from other species.
- Learning: If you put in honest effort, you will actually end up learning something!
- Marks: If you put in honest effort, I can guarantee that you will do better in the exam. Because several concepts will automatically become clear to you.
- Deterrent: Honestly I do not want to waste time trying to locate cheating. Nor is it easy. BUT if we find evidence of cheating, there will be a straight reduction in grade by 2 (A->B etc.).

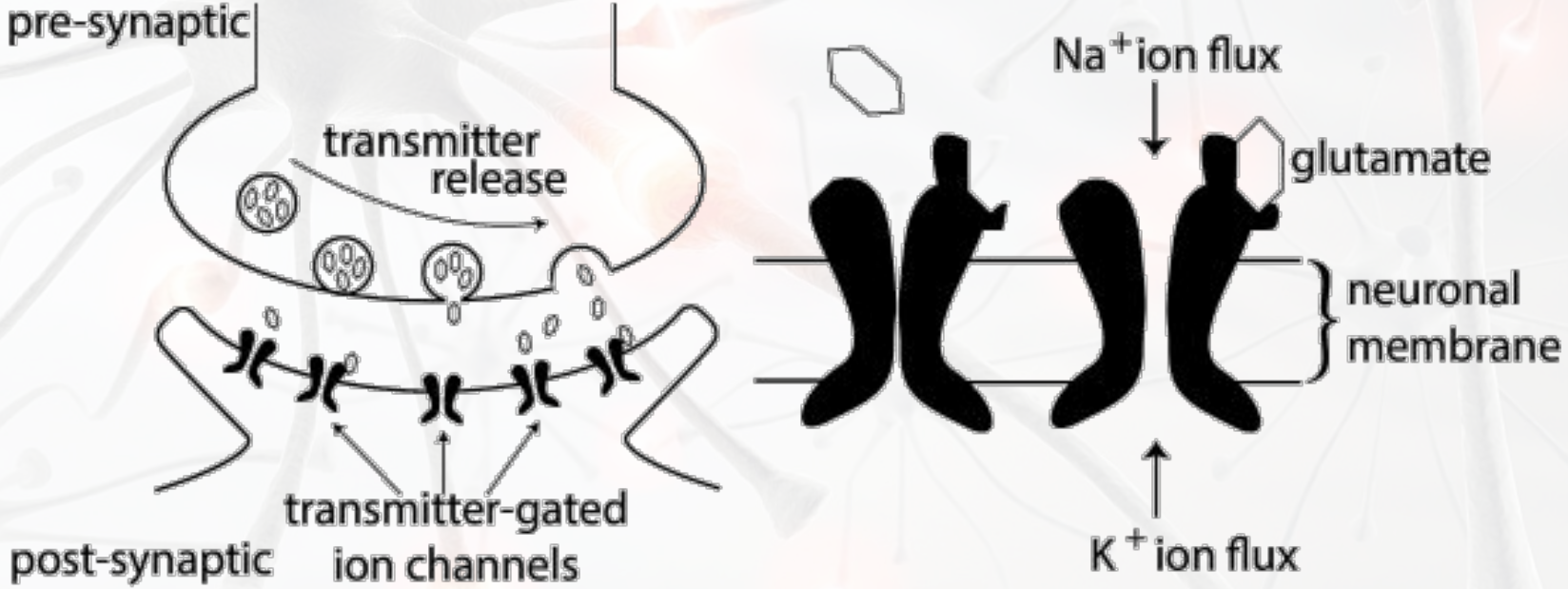
  You can decide whether you want to take the risk or not.

Grading of assignments

- The assignments are out of 5 marks
- You are allowed (and encouraged) to discuss with friends on how to solve them
- Ultimately you should write your own code / give your own solution without blind copy-pasting
- Remember the purpose – concepts should become clear. After doing the assignment, you will SELF GRADE out of 5 whether you got conceptual clarity or not!
- We will evaluate 10% of randomly selected assignments – clarity of concepts should be reflected in the content
- Tuesday class: Assignment discussion – make sure you have tried it on your own to get the most out of the class

Dendrites and synapses

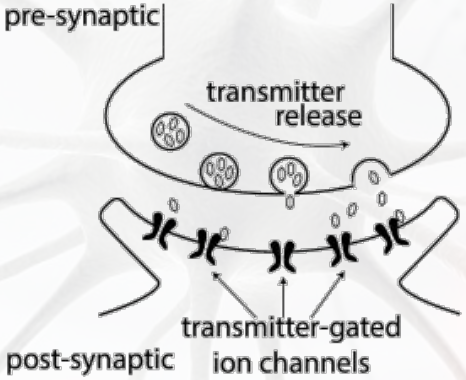
More ion channels at synapses!

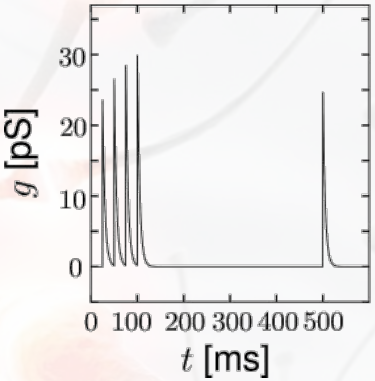


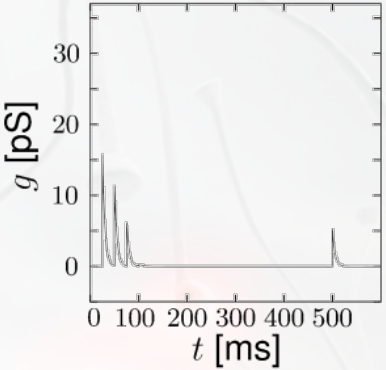
Modeling synaptic ion channels

- We have seen voltage activated and calcium activated ion channels
- Third type: transmitter activated ion channels
- Model ion flow through t-channels using a time-dependent synaptic conductivity, $g_{syn}(t)$
- Synaptic current given by: $I_{\mathrm{syn}}(t) = g_{\mathrm{syn}}(t)\ (u(t) - E_{\mathrm{syn}})$
- Different g_{syn} and E_{syn} for modelling different types of neurotransmitters
- For inhibitory synapses E_{syn} is usually set to −75 mV, for excitatory synapses $E_{syn} \approx 0$
- How does the conductivity depend on time? $g_{\mathrm{syn}}(t) = \sum_f \bar{g}_{\mathrm{syn}}\, \mathrm{e}^{-(t-t^{(f)})/\tau}\, \Theta\left(t - t^{(f)}\right)$
- More detailed model: $g_{\mathrm{syn}}(t) = \sum_f \bar{g}_{\mathrm{syn}} \left[1 - \mathrm{e}^{-(t-t^{(f)})/\tau_{\mathrm{rise}}}\right] \left[a\, \mathrm{e}^{-(t-t^{(f)})/\tau_{\mathrm{fast}}} + (1-a)\, \mathrm{e}^{-(t-t^{(f)})/\tau_{\mathrm{slow}}}\right] \Theta\left(t - t^{(f)}\right)$
- What happens when an inhibitory signal arrives at resting state?
- What happens when it arrives at an already hyperpolarized neuron?

Synaptic facilitation and depression





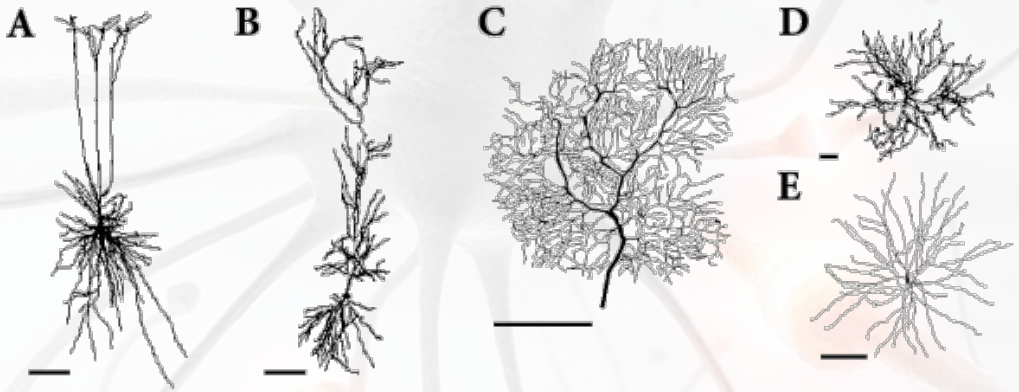


- When an AP arrives along the axon, only a fraction of the presynaptic sites release neurotransmitter: P_{rel}
- A second AP arriving will cause more (or less) sites to release the neurotransmitter, leading to facilitation (or depression)
- These are a form of very short term memory
- Match the following equations with facilitation or depression:

$$\frac{dP_{\text{rel}}}{dt} = -\frac{P_{\text{rel}} - P_0}{\tau_P} + f_F\,(1 - P_{\text{rel}})\,\sum_f \delta\left(t - t^f\right)$$

$$\frac{dP_{\text{rel}}}{dt} = -\frac{P_{\text{rel}} - P_0}{\tau_P} - f_D\,P_{\text{rel}}\sum_f \delta\left(t - t^f\right)$$

Modeling signal transmission in dendrites and axons

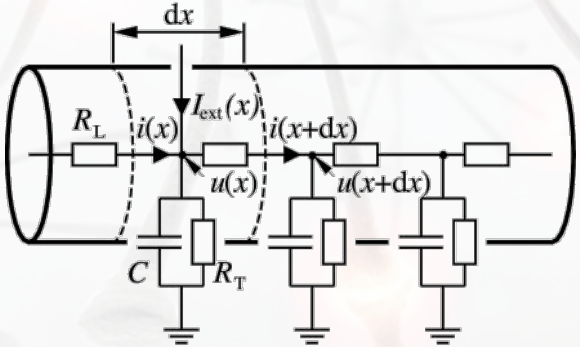


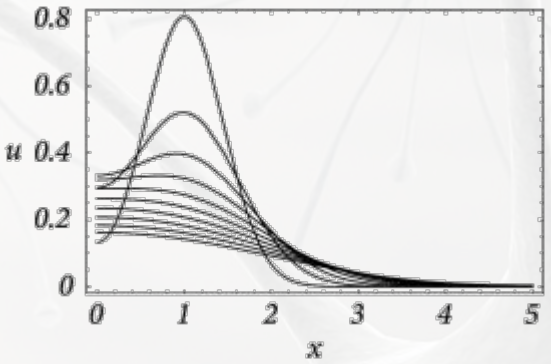
$$\frac{d^2}{dx^2}u(t,x) = cr_L \frac{d}{dt}u(t,x) + r_L \sum_{ion} i_{ion}(t,x) - r_L i^{ext}(t,x)$$

$\sum_{ion} i_{ion}(t,x) = leak$ passive dendrite

$\sum_{ion} i_{ion}(t,x) = Ca, Na, \ldots$ active dendrite

$\sum_{ion} i_{ion}(t,x) = Na, K, \ldots$ axon





The HH model is rather too complex, isn't it?

- What makes it so difficult to understand and analyze HH?
- If we have *n* ion channels and each have both activation and deactivation, what is the dimensionality of the HH-type model?

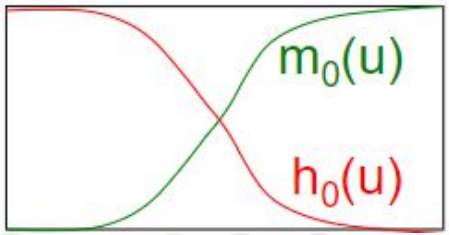
Can we reduce the dimensionality in the HH model?

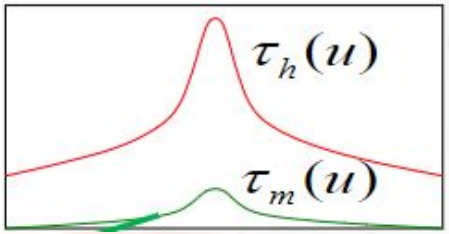
Two principles that we will use:

- Separation of timescales
- Exploit similarities and correlations

What is separation of timescales?

$$C\frac{\mathrm{d}u}{\mathrm{d}t} = g_{\mathrm{Na}}\, m^3 h\, (u - E_{\mathrm{Na}}) + g_{\mathrm{K}}\, n^4\, (u - E_{\mathrm{K}}) + g_L\, (u - E_L)$$





$$\frac{dm}{dt} = -\frac{m - m_0(u)}{\tau_m(u)}$$
$$\frac{dh}{dt} = -\frac{h - h_0(u)}{\tau_h(u)}$$
$$\frac{dn}{dt} = -\frac{n - n_0(u)}{\tau_n(u)}$$

- Dynamics of *m* is fast! $\longrightarrow$ $m(t) = m_0(u(t))$

Two coupled differential equations

$$\tau_1 \frac{dx}{dt} = -x + h(y)$$
$$\tau_2 \frac{dy}{dt} = f(y) + g(x)$$

Separation of time scales

$$\tau_1 \ll \tau_2 \rightarrow x = h(y)$$

Reduced 1-dimensional system

$$\tau_2 \frac{dy}{dt} = f(y) + g(h(y))$$

So now we are left with a 3D system

$$C\frac{\mathrm{d}u}{\mathrm{d}t} = g_{\mathrm{Na}}\, m^3 h\, (u - E_{\mathrm{Na}}) + g_{\mathrm{K}}\, n^4\, (u - E_{\mathrm{K}}) + g_L\, (u - E_L)$$

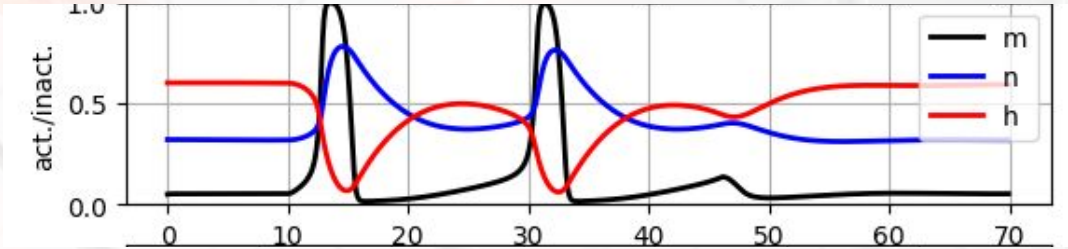
$$\frac{dh}{dt} = -\frac{h - h_0(u)}{\tau_h(u)}$$

$$\frac{dn}{dt} = -\frac{n - n_0(u)}{\tau_n(u)}$$

Can we simplify further by exploiting correlations?

- Dynamics of *h* and *n* are similar

$$1 - h(t) = a\, n(t) = w(t)$$



$$\frac{dw}{dt} = -\frac{w - w_0(u)}{\tau_{eff}(u)} \qquad C\frac{du}{dt} = -g_{Na}\, m_0(u)^3 (1 - w)(u - E_{Na}) - g_K [\frac{w}{a}]^4 (u - E_K) - g_l (u - E_l)$$

When can we separate the timescales?

We start with two equations

$$\tau_1 \frac{dx}{dt} = -x + y + I(t)$$

$$\tau_2 \frac{dy}{dt} = -y + x^2 + A$$

[ ] If $\tau_1 \ll \tau_2$ then the system can be reduded to

$$\tau_2 \frac{dy}{dt} = -y + [y + I(t)]^2 + A$$

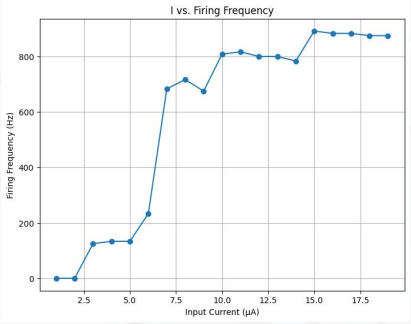
[ ] If $\tau_2 \ll \tau_1$ then the system can be reduded to

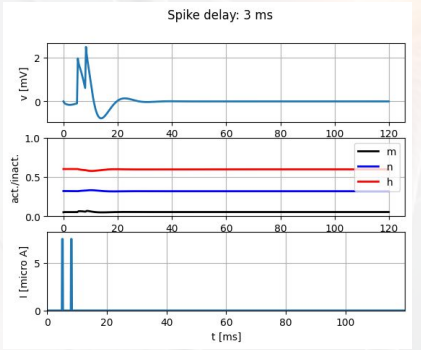
$$\tau_1 \frac{dx}{dt} = -x + x^2 + A + I(t)$$

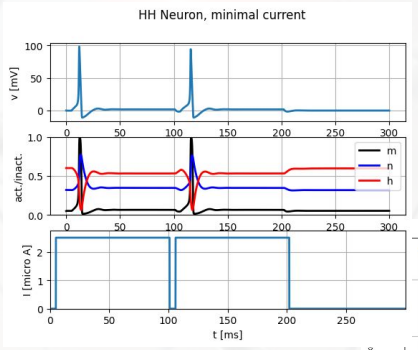
[ ] None of the above is correct.

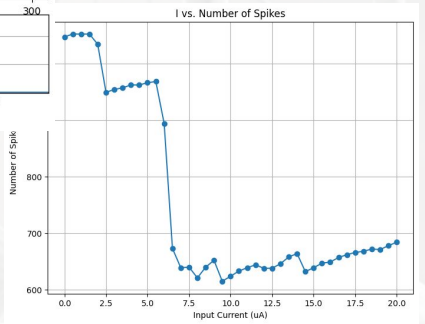
Assignment 1 pitfalls!

- Many have not started yet. Clearly they cannot give themselves 4/5 marks!
- Time to first spike – some of you have used time to cross threshold as a proxy. This may not be a good idea, your threshold is arbitrary
- Firing rate computation: #spikes/interval is not a good estimate. #sustained spikes/interval is better. Even better is 1/<DT>
- Current pulse generation: Note that the input_factory function accepts only integers for t_start and t_end, so you will have to change units!
- Using max(vm) is not sufficient to detect a spike
- Refractoriness is relevant with SHORT pulses









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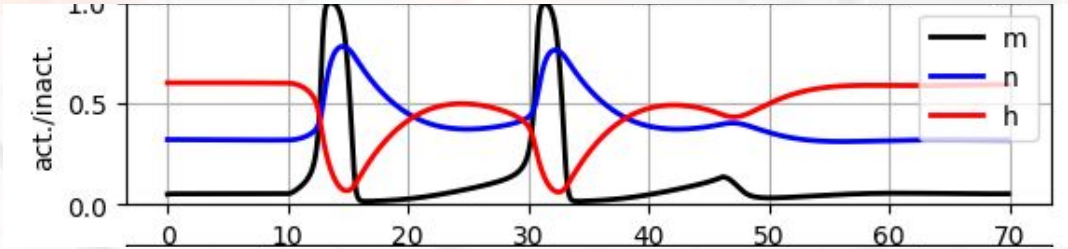

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How do we analyze our 2D system now?

$$\frac{dw}{dt} = -\frac{w - w_0(u)}{\tau_{eff}(u)} \qquad C\frac{du}{dt} = -g_{Na}\, m_0(u)^3 (1-w)(u - E_{Na}) - g_K[\frac{w}{a}]^4 (u - E_K) - g_l(u - E_l)$$

stimulus

$$\tau \frac{du}{dt} = F(u,w) + RI(t)$$

$$\tau_w \frac{dw}{dt} = G(u,w)$$

$$\tau \frac{du}{dt} = F(u,w) + RI(t)$$

$$= u - \frac{1}{3}u^3 - w + RI(t)$$

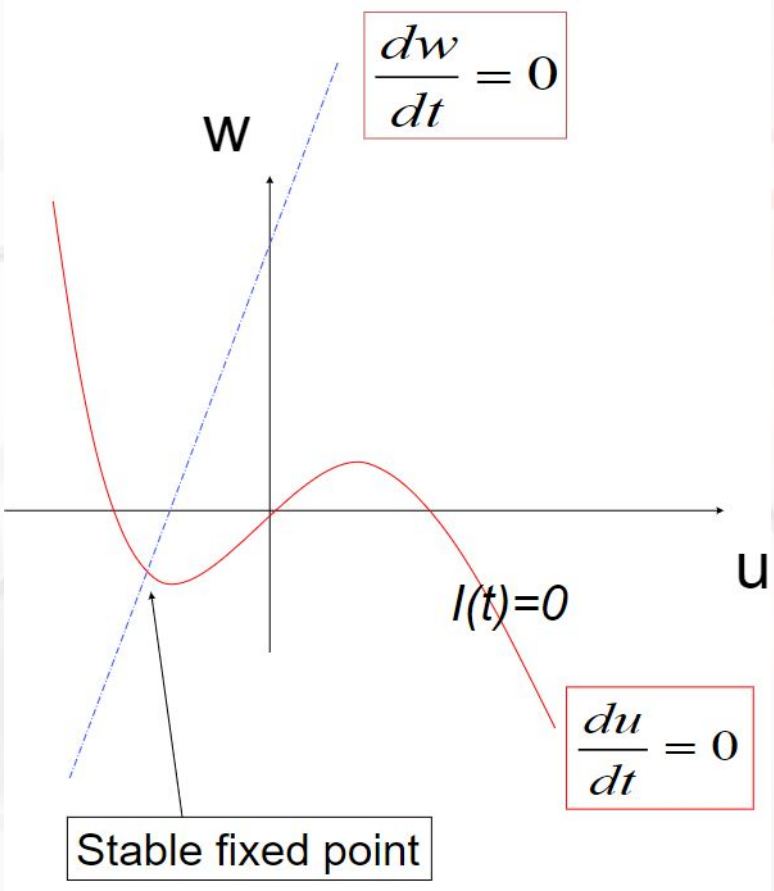
$$\tau_w \frac{dw}{dt} = G(u,w) = b_0 + b_1 u - w$$

Phase plane analysis

Also called state space analysis

Flow on and between nullclines

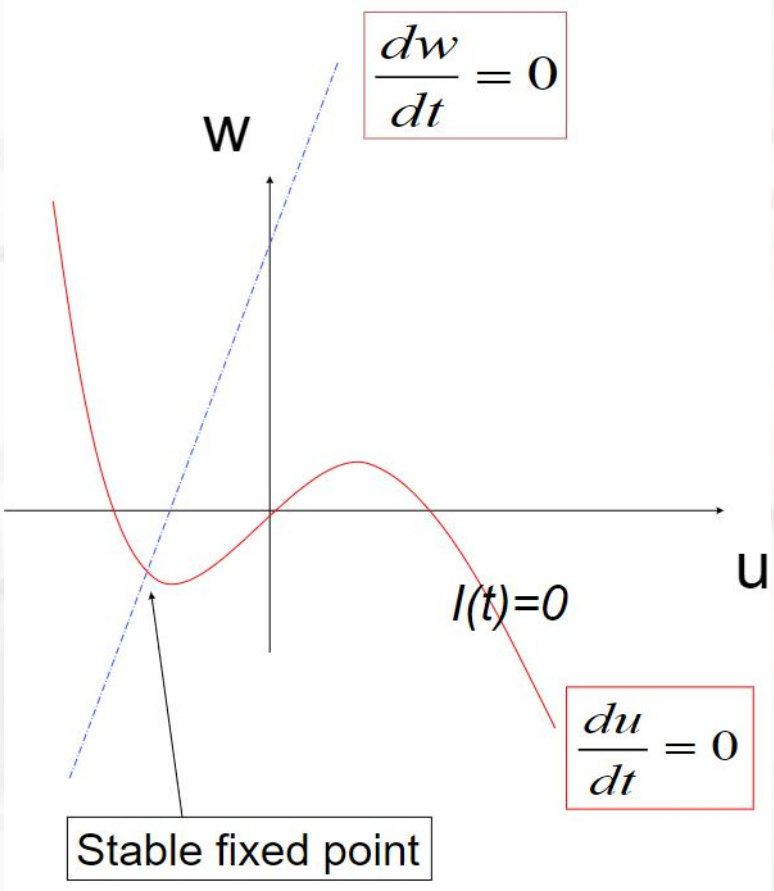
$$\tau \frac{du}{dt} = F(u,w) + RI(t)$$
$$= u - \frac{1}{3}u^3 - w + RI(t)$$
$$\tau_w \frac{dw}{dt} = G(u,w) = b_0 + b_1 u - w$$



- What is a nullcline?
- On the u-nullcline, flow arrows are always
  - Vertical
  - Vertically upwards
  - Vertically downwards
  - Horizontal
  - Horizontal leftwards
  - Horizontal rightwards
- What about the w-nullcline?
- What is a fixed point?
- What is a stable or unstable fixed point?

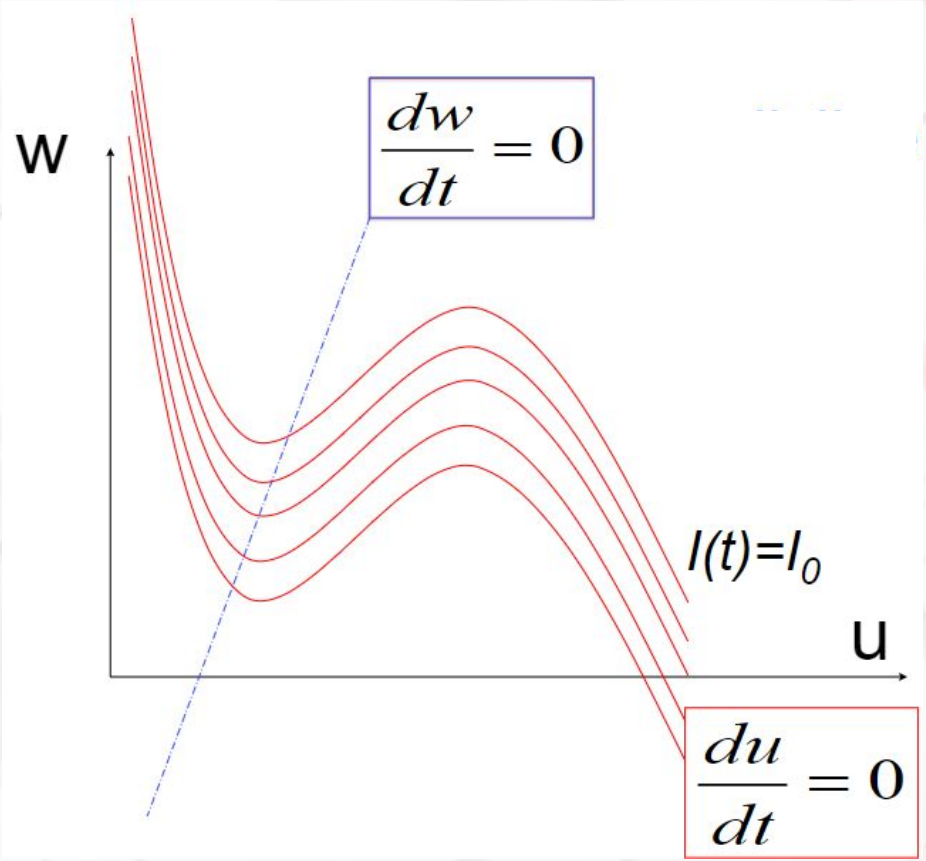
FitzHugh-Nagumo Model

$$\tau \frac{du}{dt} = F(u,w) + RI(t)$$
$$= u - \frac{1}{3}u^3 - w + RI(t)$$
$$\tau_w \frac{dw}{dt} = G(u,w) = b_0 + b_1 u - w$$



- How does the system typically behave?
- How does it depend on initial conditions?
- When is a fixed point stable?
- What happens with unstable fixed points?
- What happens when you give a short pulse of current to this system?
- What happens when you apply a constant current input to the system?
- Changing relative timescales
- How can we study inhibitory rebound?

FitzHugh-Nagumo Model



FHN under a constant applied current:

- Fixed point moves
- Stability changes

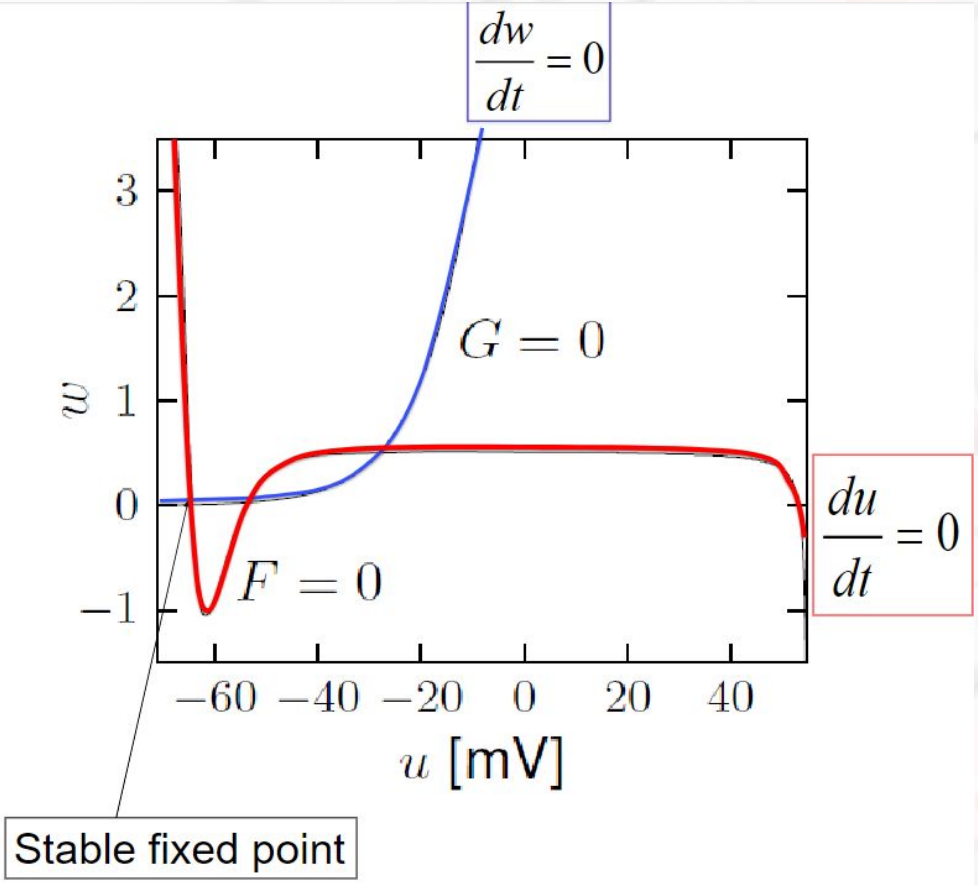
Limit cycles

Threshold

Delayed spikes vs reduced amplitudes

Inhibitory rebound

Simplified HH model phase plane



Non-linear integrate and fire models

The origin of non-linear integrate and fire models

stimulus

$$\tau \frac{du}{dt} = F(u,w) + RI(t)$$

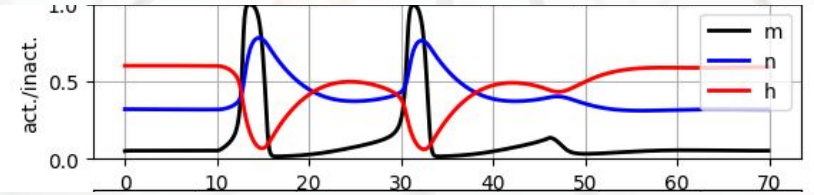
$$\tau_w \frac{dw}{dt} = G(u,w)$$

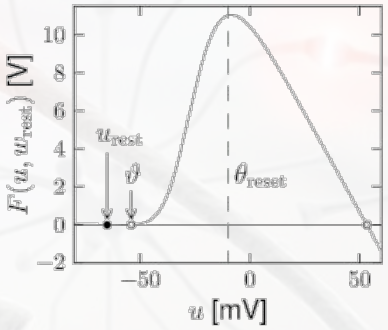
$$\frac{\mathrm{d}u}{\mathrm{d}t} = F(u,w) + I$$

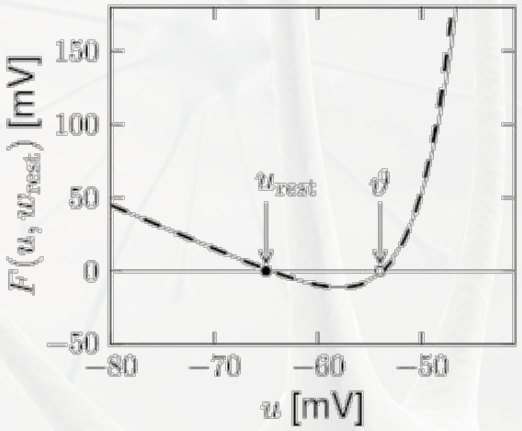
$$\frac{\mathrm{d}w}{\mathrm{d}t} = \epsilon\, G(u,w)$$

$(\epsilon \ll 1)$

$$\frac{\mathrm{d}u}{\mathrm{d}t} = F(u, w_{\mathrm{rest}}) + I = f(u) + I$$



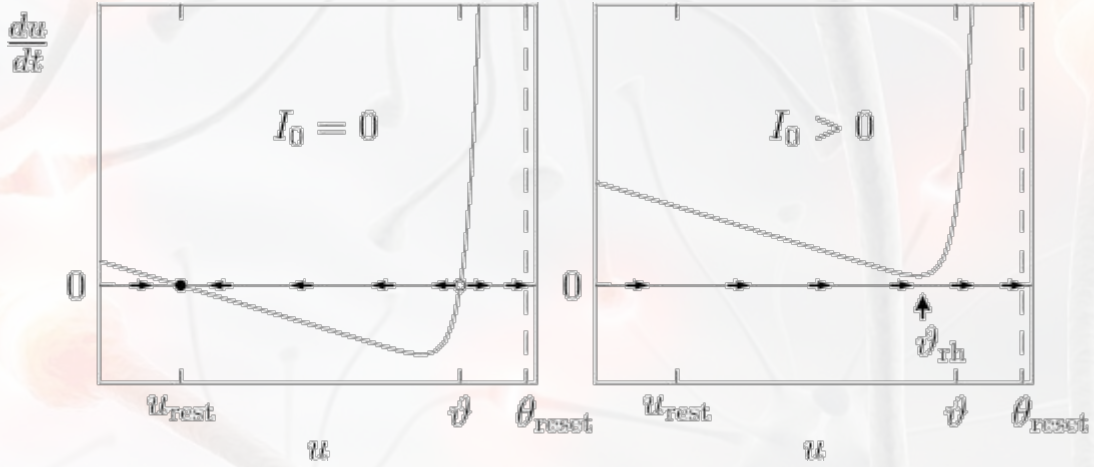




$$C\frac{\mathrm{d}\ u}{\mathrm{d}\ t} = -g_{\mathrm{Na}}[m_0\,(u)]^3\, h_{\mathrm{rest}}\ (u - E_{\mathrm{Na}}) - g_{\mathrm{K}}\,(n_{\mathrm{rest}})^4\ (u - E_{\mathrm{K}}) - g_L\ (u - E_L) + I$$

General form and working principle of NLIF models

$$\tau \frac{\mathrm{d}}{\mathrm{d}t} u = f(u) + R(u)\, I$$



- Voltage threshold?
- What is the stability of the two fixed points?
- Current threshold?
- Rheobase voltage
- Specific form: Exponential Integrate and Fire

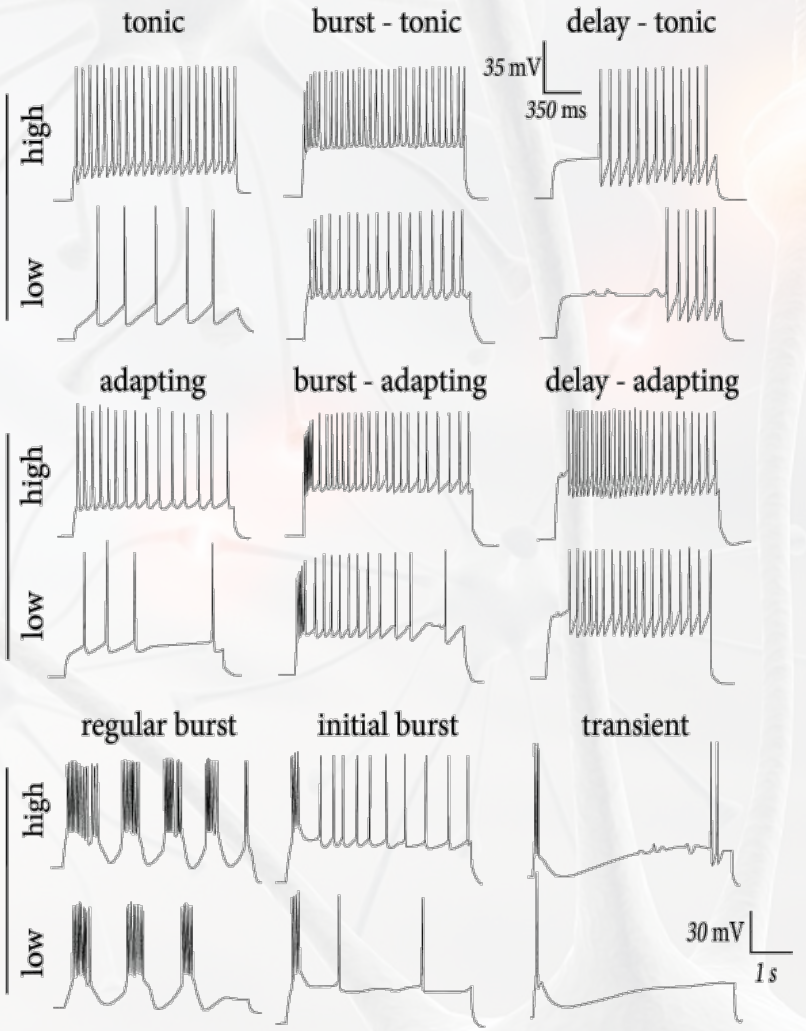
$$\tau \frac{\mathrm{d}}{\mathrm{d}t} u = -(u - u_{\mathrm{rest}}) + \Delta_T \exp\left(\frac{u - \vartheta_{rh}}{\Delta_T}\right) + R\, I$$

Adaptation in the NLIF model

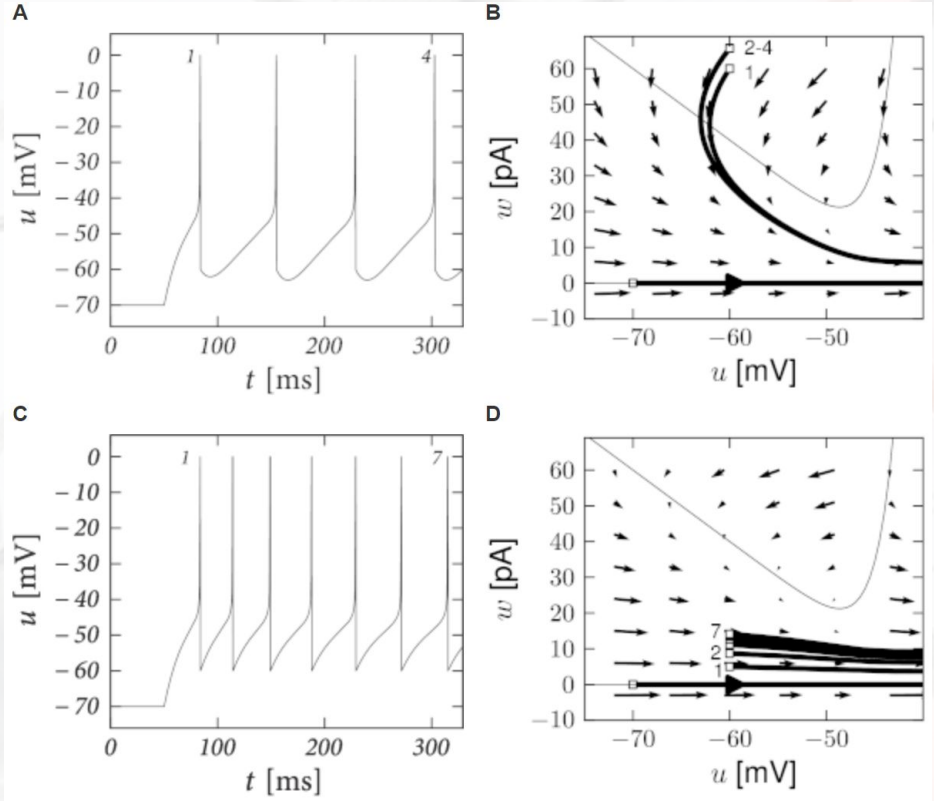
$$\tau_m \frac{\mathrm{d}u}{\mathrm{d}t} = -(u - u_{\mathrm{rest}}) + \Delta_T \exp\left(\frac{u - \vartheta_{rh}}{\Delta_T}\right) - R\,w + R\,I(t)$$

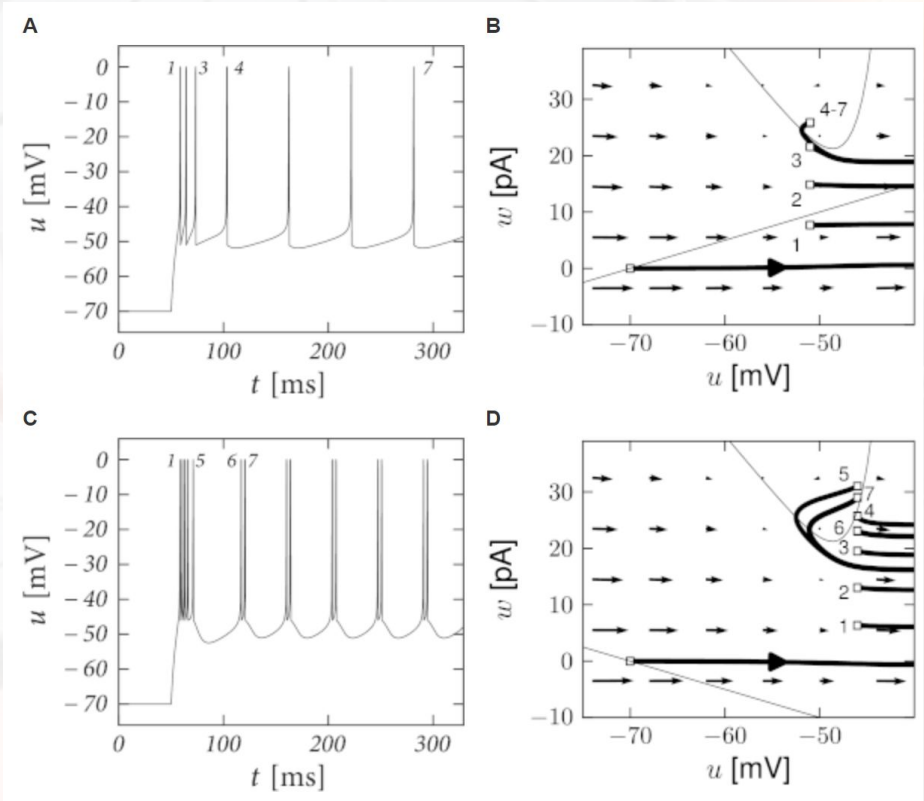
$$\tau_w \frac{\mathrm{d}w}{\mathrm{d}t} = a\,(u - u_{\mathrm{rest}}) - w + b\tau_w \sum_{t^{(f)}} \delta\left(t - t^{(f)}\right) .$$

- Add a time-varying “inhibitory current” to the original NLIF model
- Why? Remember the Ca^{2+} influx?
- Inhibitory current depends on previous spikes
- Which of τ_m and τ_k should be larger?



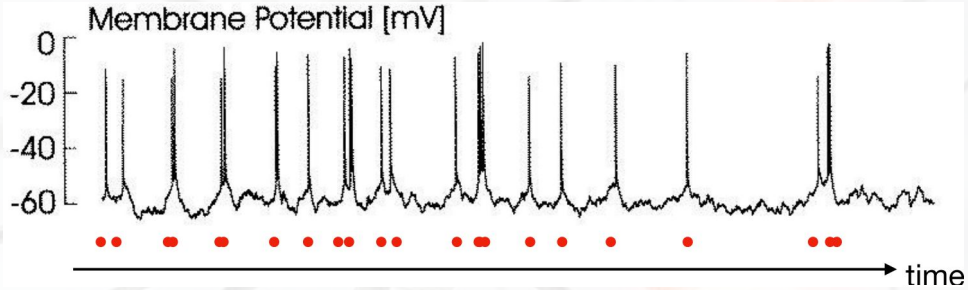
Phase plane analysis of adaptation in NLIF models





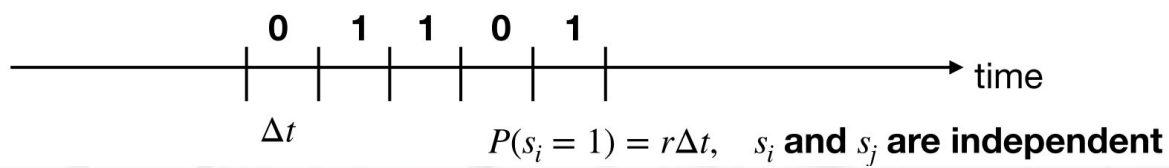
Spikes are stochastic!

Poisson neuron



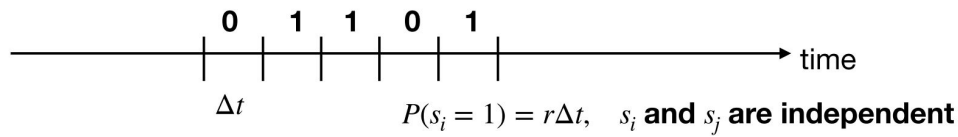
Directly model spike times, and not the underlying process!

- Poisson process – one of the simplest point processes



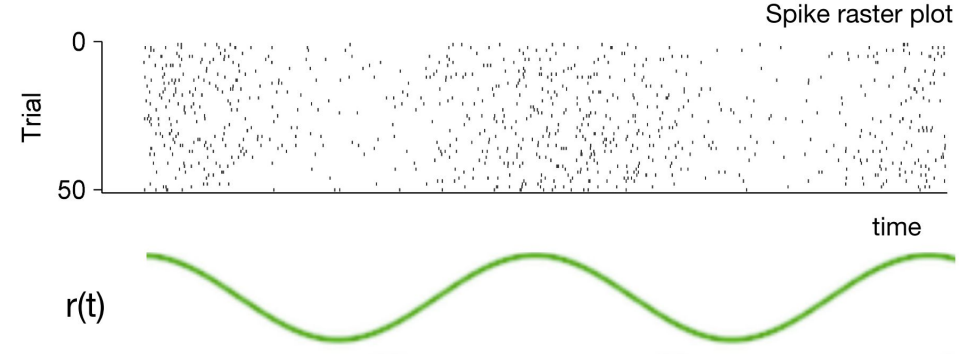
- Fixed spike probability in each interval – constant ‘firing rate’
- Interspike interval (ISI) follows what distribution?
- Exponential!

Poisson neuron with time varying rate



$P(s_i = 1) = r\Delta t$, s_i **and** s_j **are independent**

$r \rightarrow r(t)$

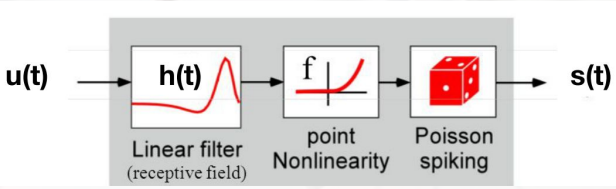


Linear-Nonlinear Poisson (LNP) model

- How does the firing rate depend on the stimulus?

$$r(t) = f(h(t) * u(t)) = f\left(\int_0^T h(\tau)u(t-\tau)d\tau\right)$$

- *f* is some non-linear scalar function
- Non-linearity required to avoid negative firing rates
- What is the ‘linear’ part here?



- How do we find *h* and *f* experimentally?

Spike-triggered averaging (STA)

1. Use *u(t)* that is white noise (random and independent in time input)
2. For every time the neuron spikes, record the stimulus up to T steps in the past – this is the spike-triggered stimulus
3. After collecting many spike-triggered stimuli, average them to estimate *h(t)*
4. After finding *h(t)*, calculate *h(t)*u(t) = I(t)*
5. Plot spike rate, *r(t)* vs *I(t)*to estimate *f(I)*

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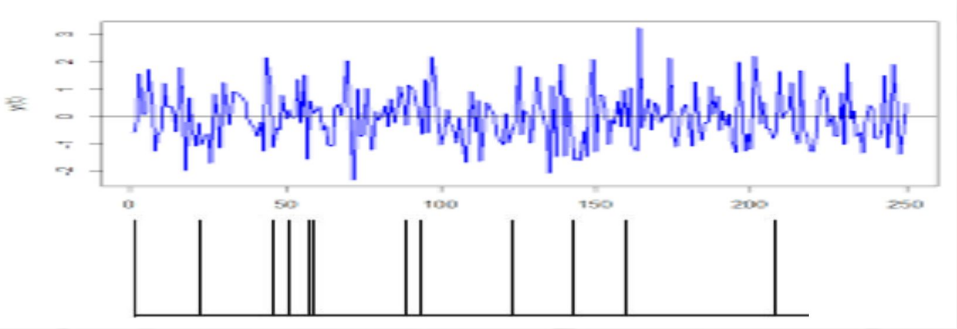

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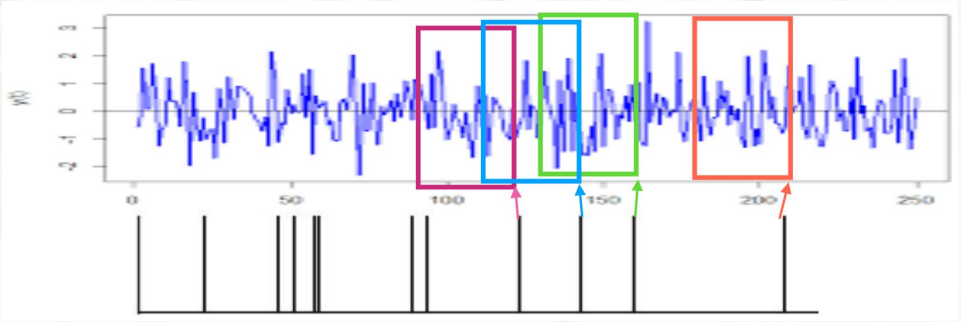

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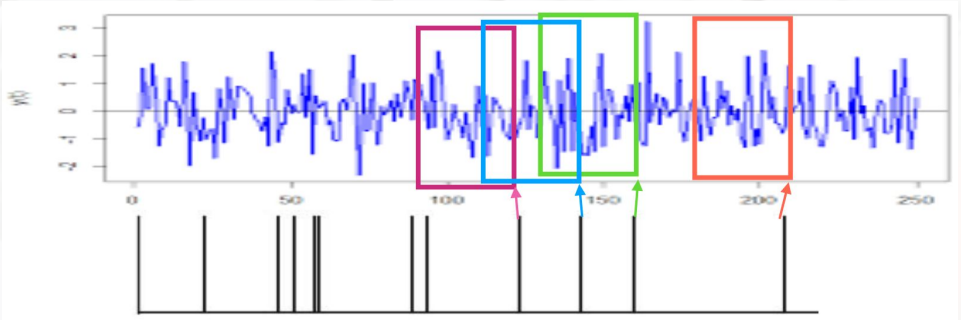

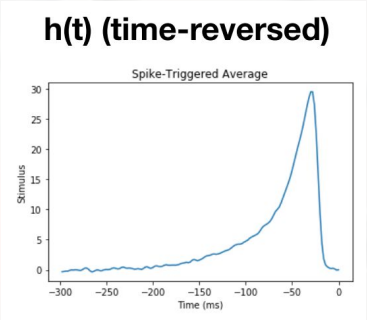

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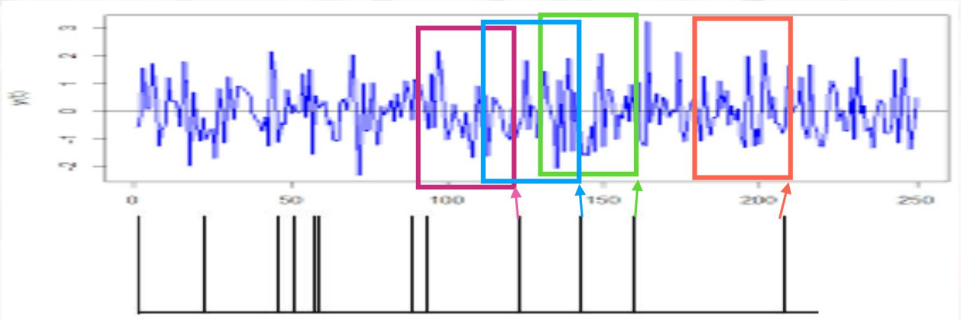

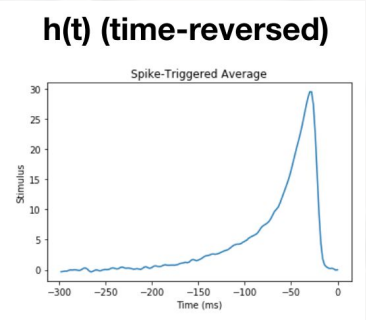

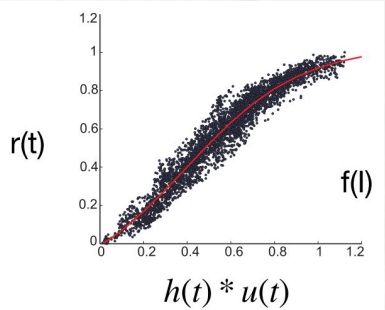

STA for 2D inputs – V1 filters