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Master degree in
Computer Science

Spiking Neural Networks

In Depth Study for Data & Computational Biology
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Oltolini Edoardo - 869124

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Biological Neurons & Simulations

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Biological Neurons & Simulations

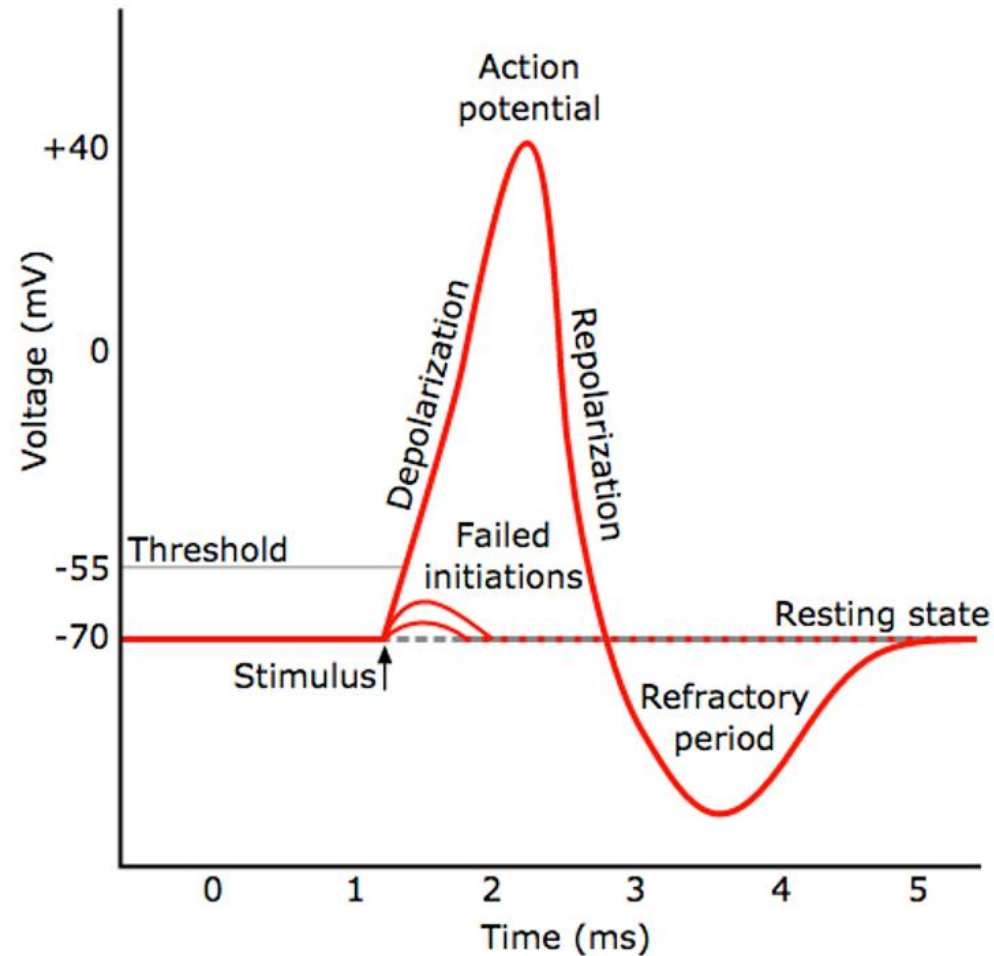
Main References:

Basic Neural Units of the Brain: Neurons, Synapses and Action Potential,
Jiawei Zhang, Information Fusion and Mining Laboratory, University of California,
Davis

**Dynamical Systems in Neuroscience: The Geometry of Excitability and
Bursting**, Eugene M. Izhikevich, The Neuroscience Institute

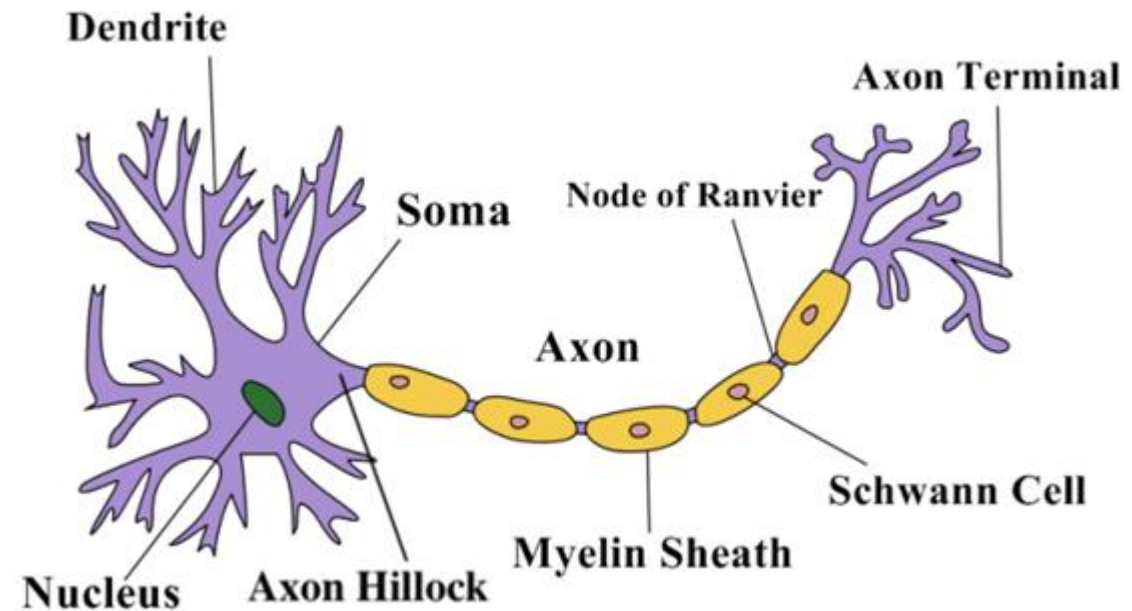
Simple Model of Spiking Neurons, Eugene M. Izhikevich, in IEEE Transactions
on Neural Networks, vol. 14, no. 6, pp. 1569-1572, Nov. 2003

Phases of a Spiking Neuron



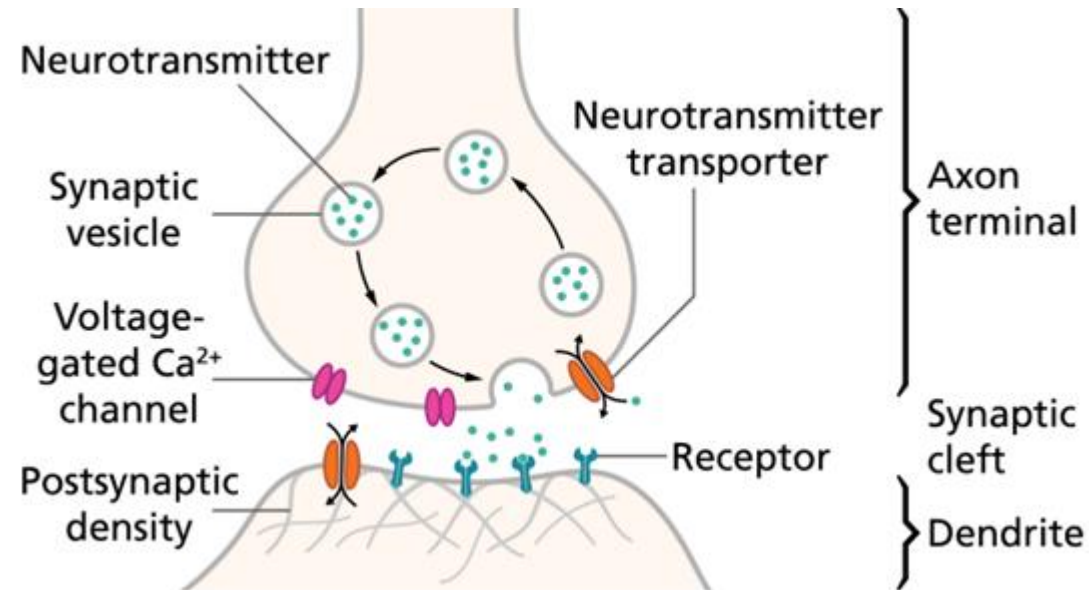
Neurons

A neuron is an electrically excitable cell that processes and transmits information by electro-chemical signaling. Each neuron may be connected to 10 thousand other neurons, passing signals to each other via 1000 trillion synaptic connections (\equiv computer with 1 trillion bits/s processor).



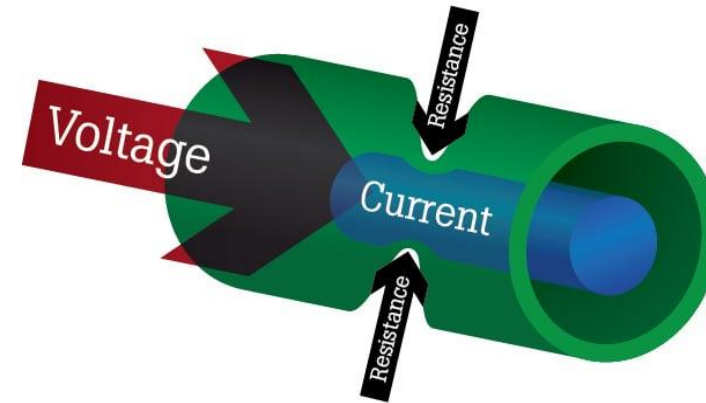
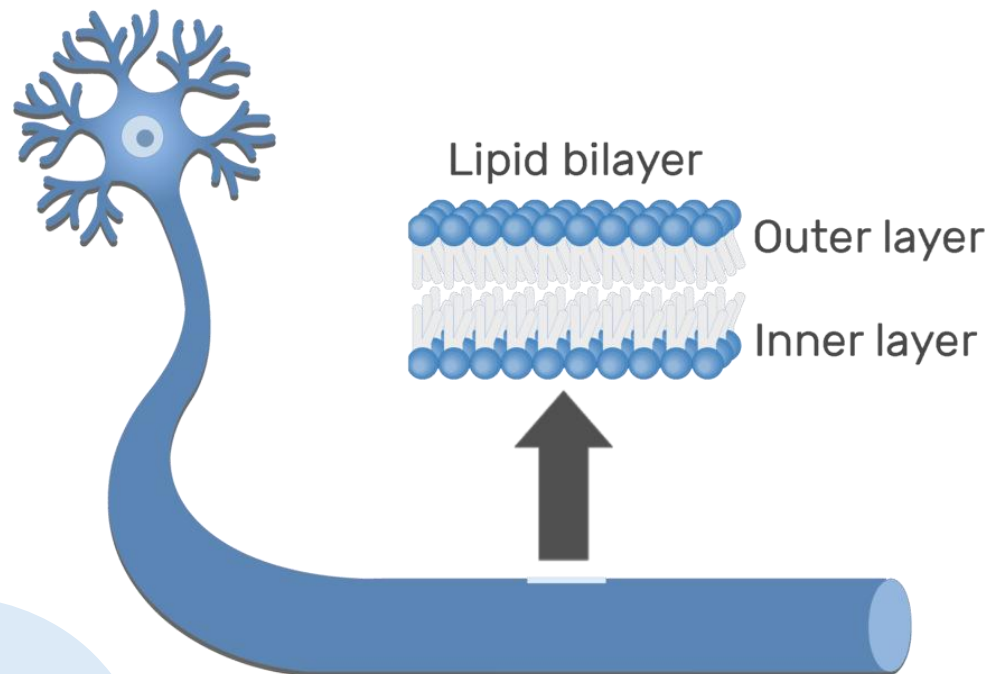
Axon Terminals & Dendrites

The process of a spike being fired and then received by another neuron takes less than 2ms



This is called a "**synapse**"

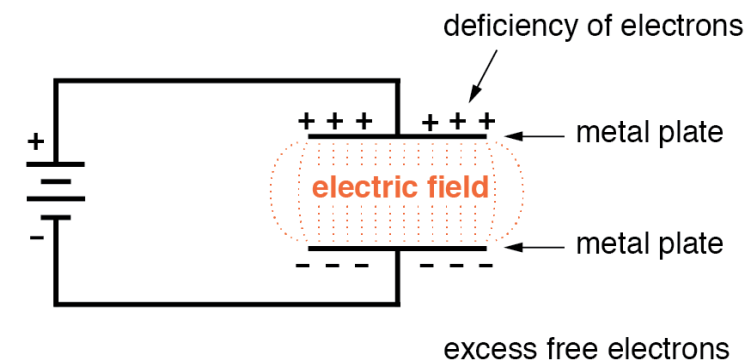
Membrane Structure



Every animal cell is enclosed in a **plasma membrane**, which has the structure of a **lipid bilayer**. This membrane has high **electrical resistivity** due to the lipid molecules.

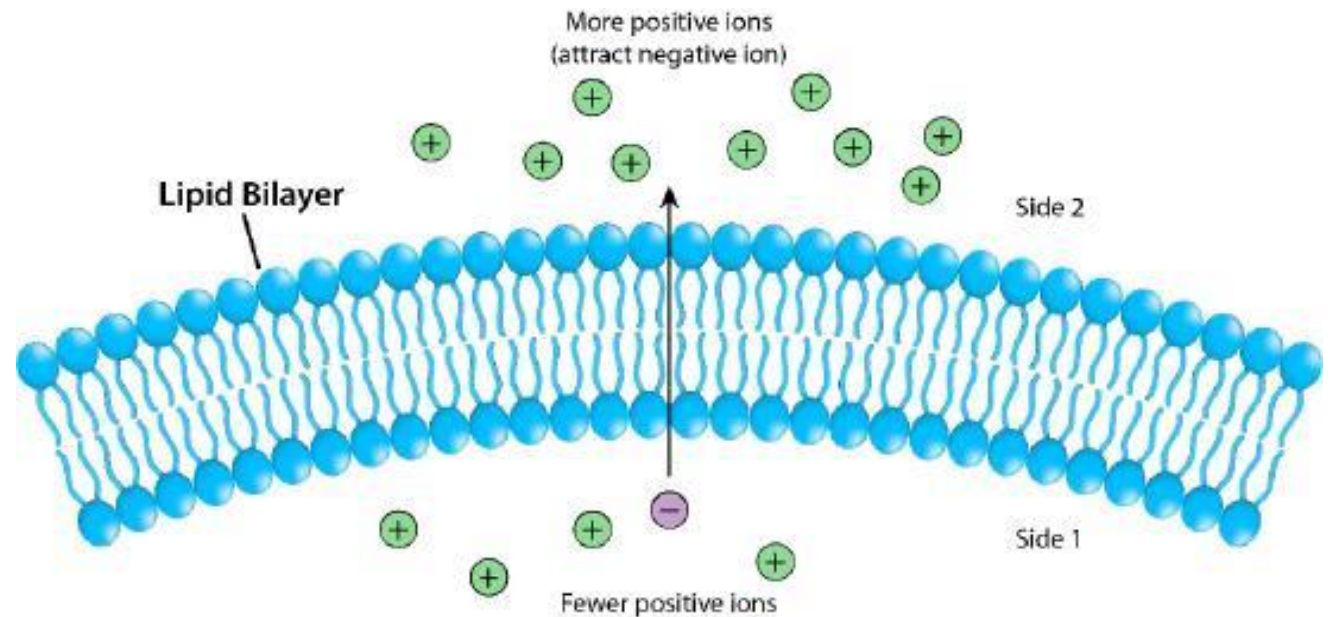
A membrane works as a combined **resistance** and **capacitance**.

Capacitance arises from the fact that the bilayer is so thin that an accumulation of charged particles on one side gives rise to an electrical force that pulls oppositely charged particles toward the other side.

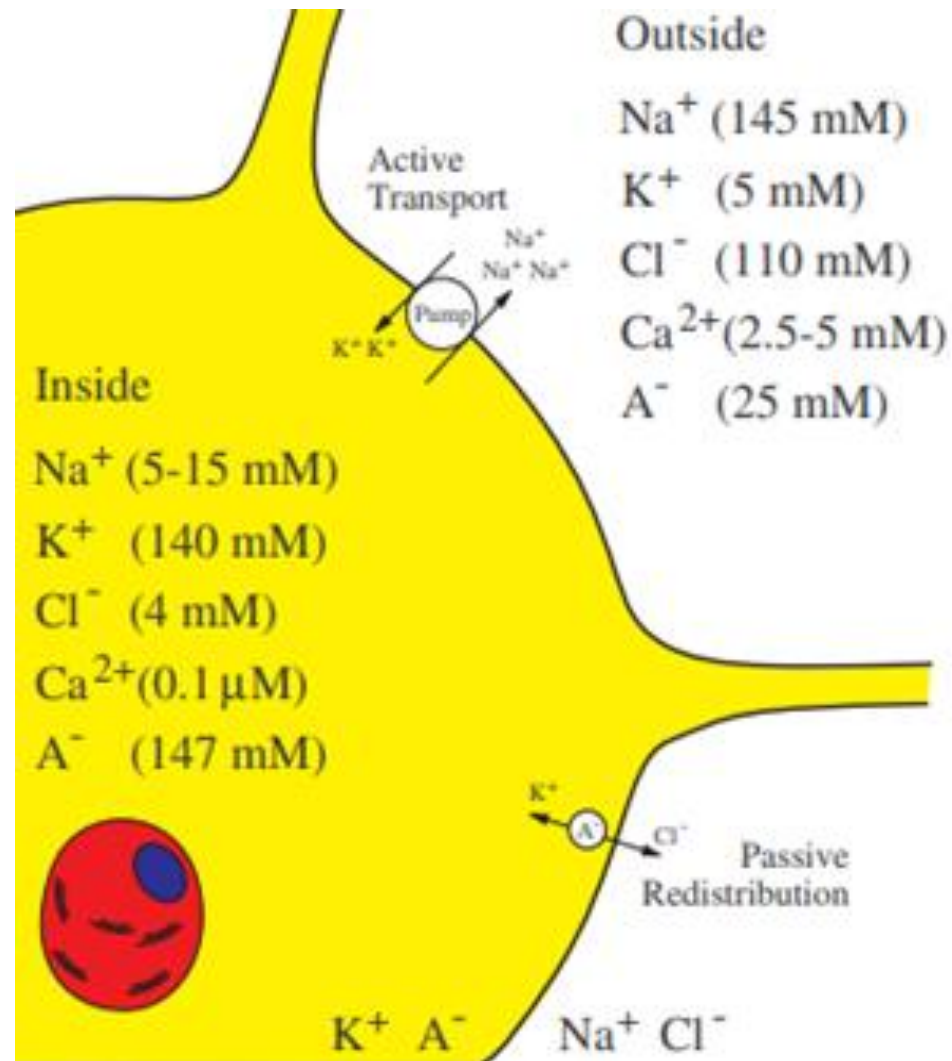


The Importance of Ions:

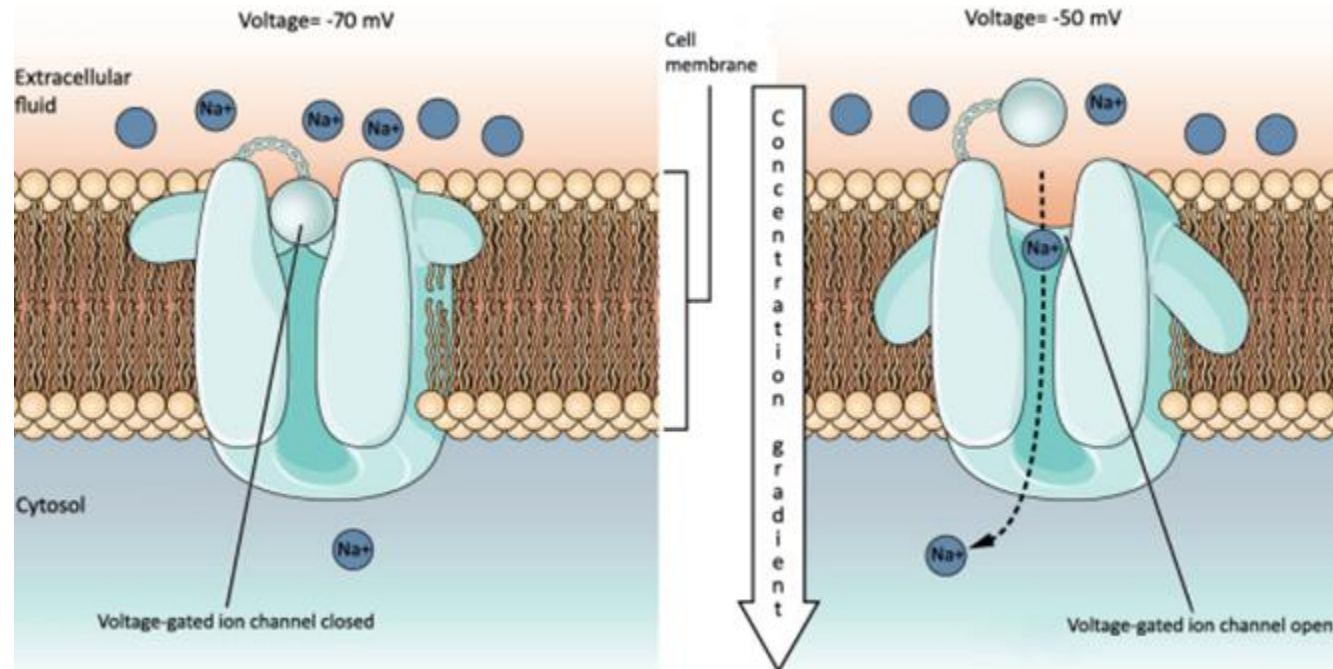
Every neuron maintains a **voltage gradient** across its membrane, due to the differences in ions of **Sodium (Na^+)**, **Potassium (K^+)**, **Chloride (Cl^-)** and **Calcium (Ca^{2+})** on the sides of the membrane. If the voltage changes significantly, an **electro-chemical** pulse is fired.



Redistribution of Ions



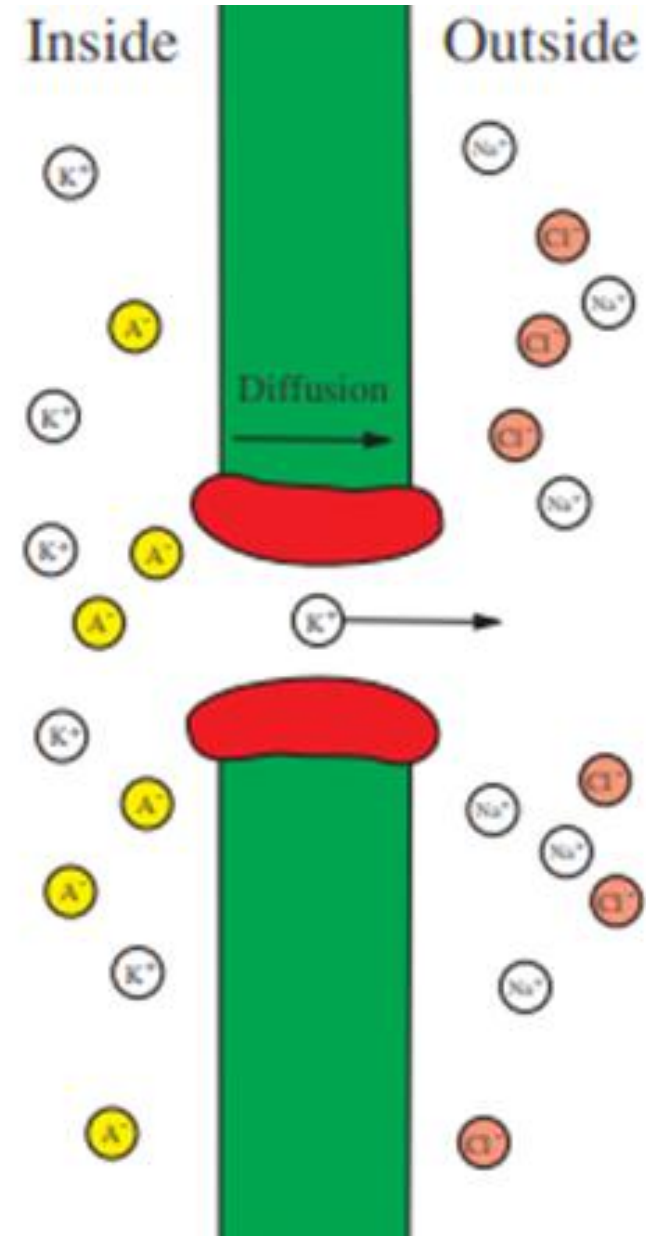
Ion Channels



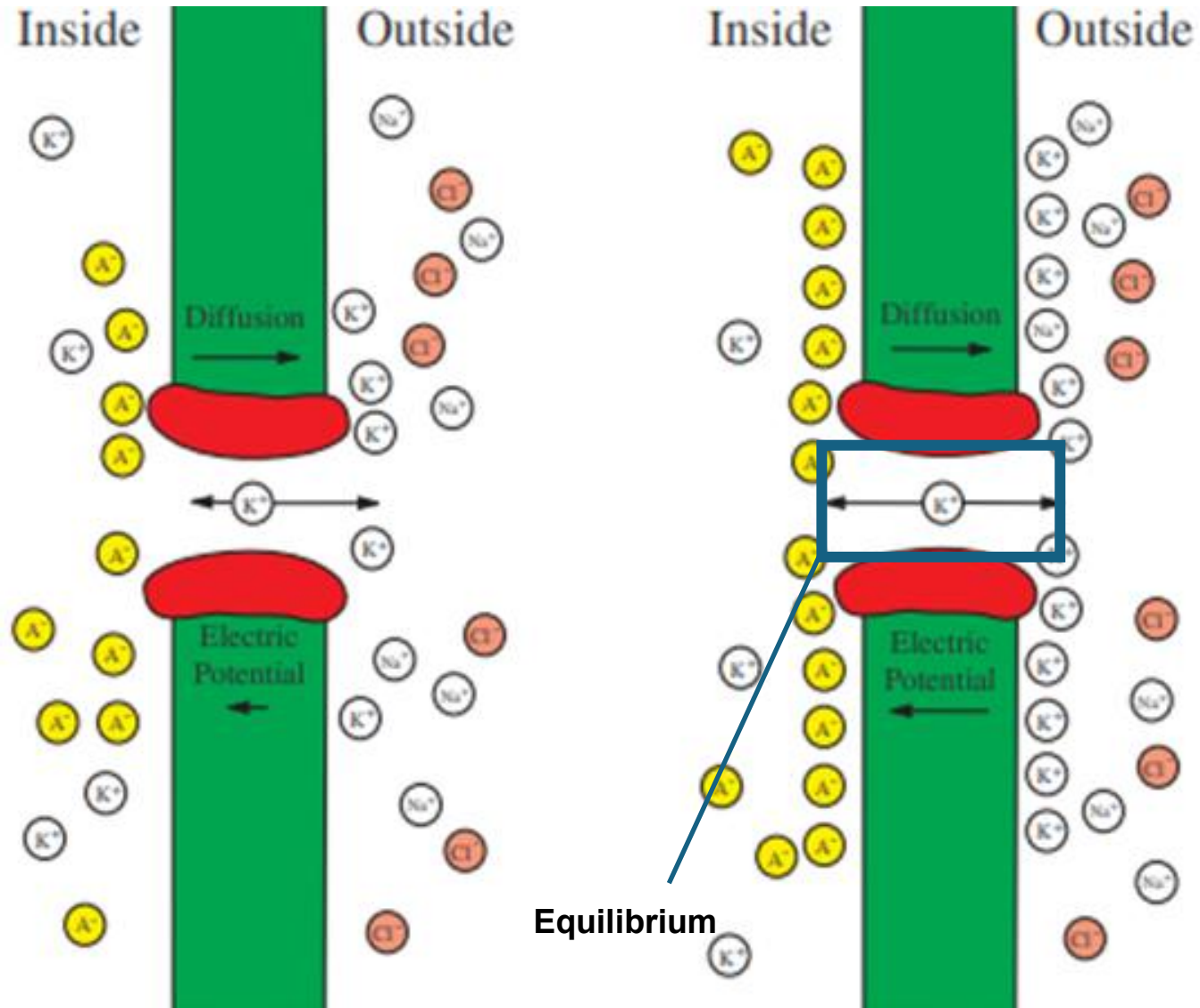
Nernst Potentials

We have **two driving forces** for ions in the channels:
concentration gradients and **electric potential** gradients.

First, the ions diffuse down the concentration gradient.



Nernst Potentials



Nernst Potentials

$$E_{\text{ion}} = \frac{RT}{zF} \ln \frac{[\text{Ion}]_{\text{out}}}{[\text{Ion}]_{\text{in}}},$$

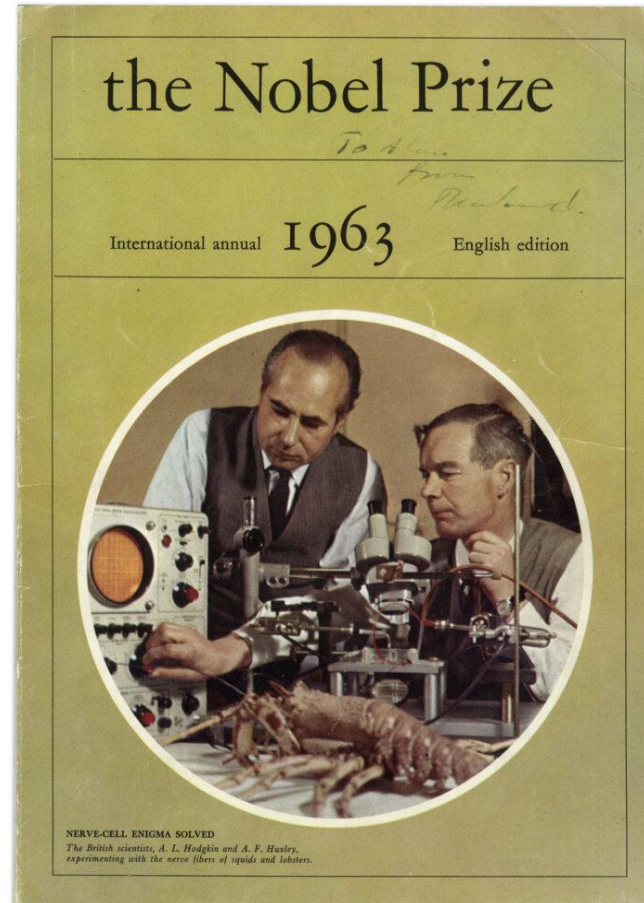
Where:

- $[\text{Ion}]_{\text{in}}$ and $[\text{Ion}]_{\text{out}}$ are concentrations of the ions inside and outside of the cell.
- R is the universal gas constant (0,315 mJ / (K°*Mol))
- T is the temperature in degrees Kelvin
- F is Faraday's constant (96480 coulombs/Mol)
- z is the valance of the ion (z = 1 for Na⁺ and K⁺, z = -1 for Cl⁻, and z = 2 for Ca²⁺).

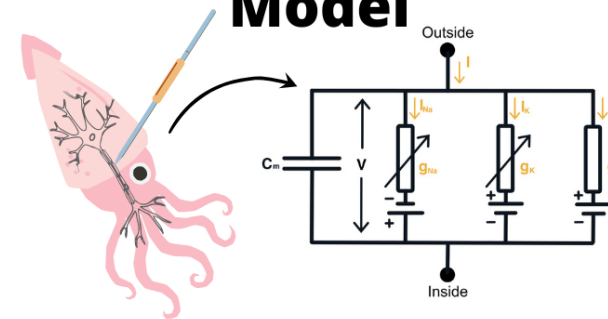
Example:

$$E_{\text{ion}} \approx 62 \log \frac{[\text{Ion}]_{\text{out}}}{[\text{Ion}]_{\text{in}}} \quad (\text{mV})$$

The Hodgkin-Huxley Model



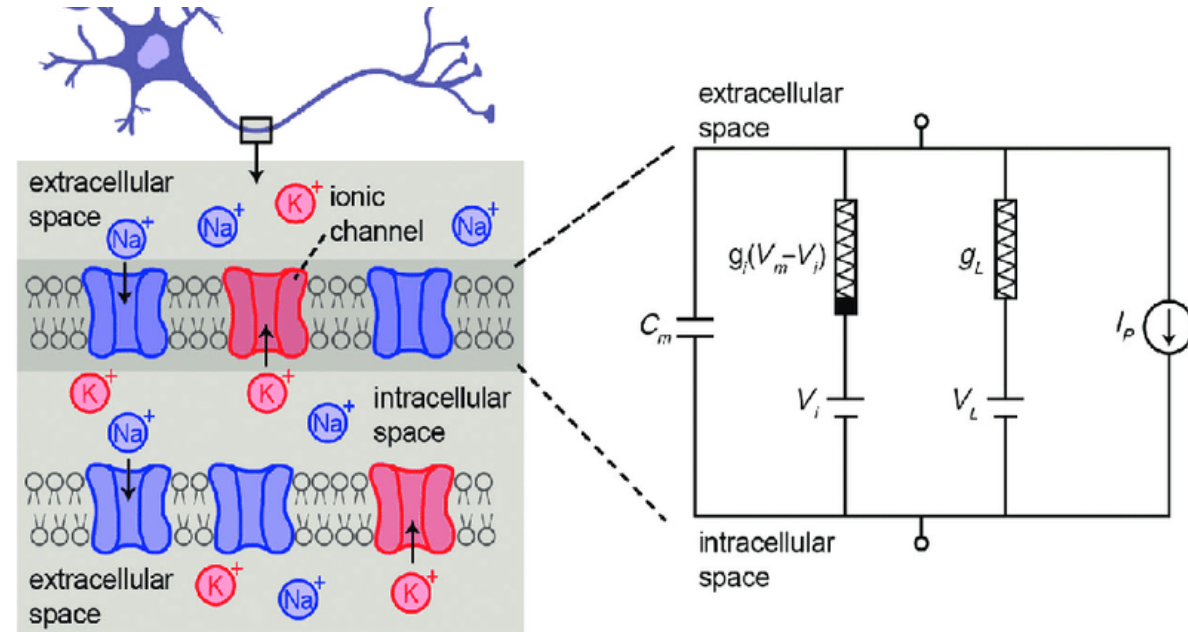
Hodgkin-Huxley Model



The Hodgkin-Huxley Model

The giant squid axons membranes carry only two currents: **transient** Na⁺ and **persistent** K⁺.

$$\frac{dQ}{dt} = C \frac{dV}{dt}$$



$$C \dot{V} = I - \overbrace{\bar{g}_K n^4 (V - E_K)}^{I_K} - \overbrace{\bar{g}_{Na} m^3 h (V - E_{Na})}^{I_{Na}} - \overbrace{g_L (V - E_L)}^{I_L}$$

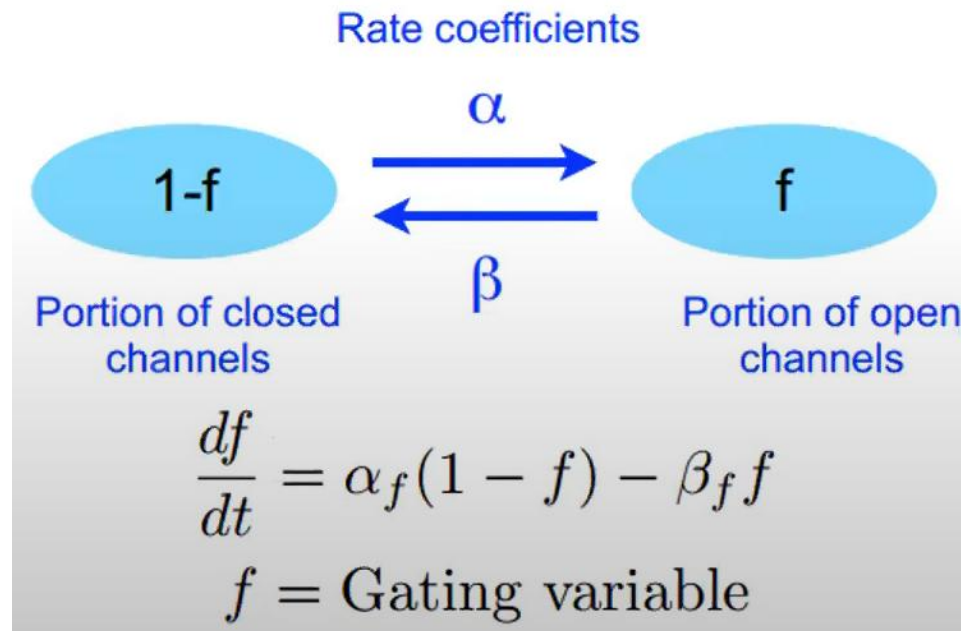
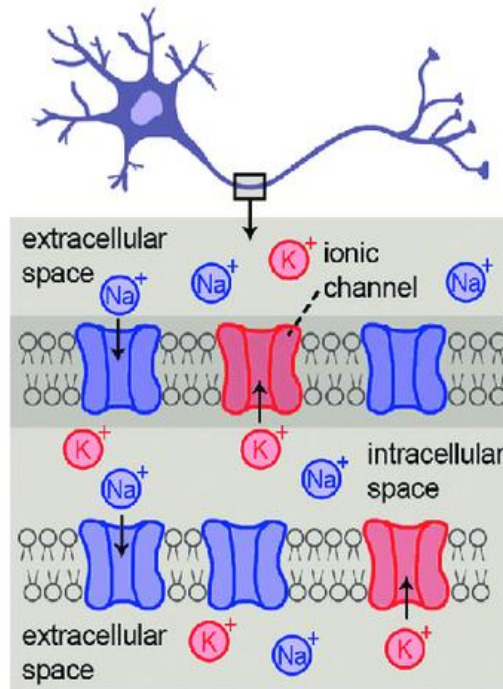
$$\dot{n} = \alpha_n(V)(1 - n) - \beta_n(V)n$$

$$\dot{m} = \alpha_m(V)(1 - m) - \beta_m(V)m$$

$$\dot{h} = \alpha_h(V)(1 - h) - \beta_h(V)h,$$

$$g = g_{MAX} * f(V, t)$$

Gating Variables



f : fraction of open channels
(gating variable, it depends on V and t)

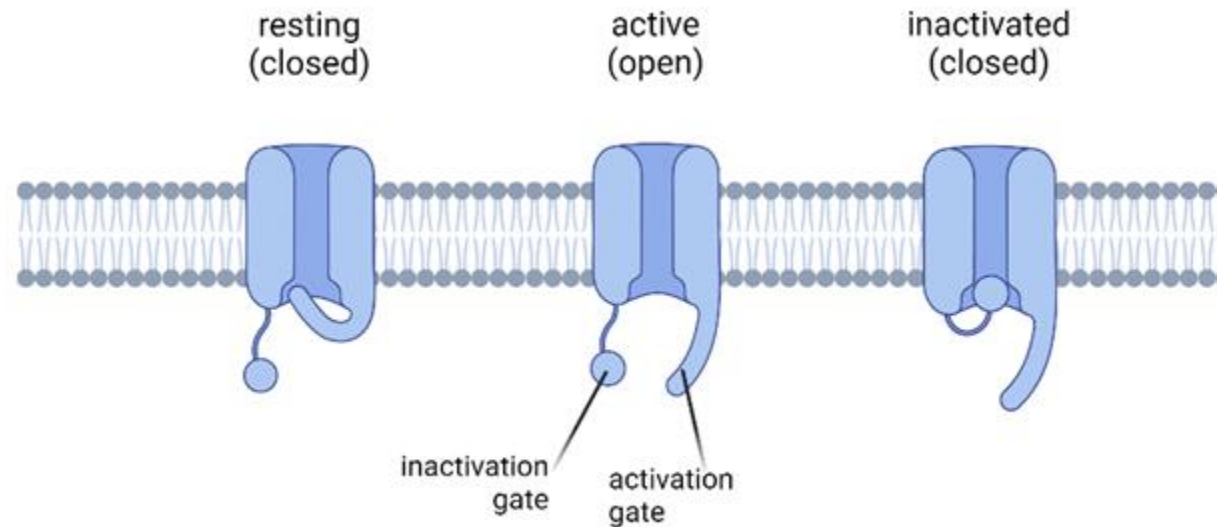
α : rate at which closed channels open (depends on V)

β : rate at which open channels close (depends on V)

Gating Variables

We're going to have **gating variables** for the potassium activation gates (n), sodium activation (m), and sodium inactivation (h).

Channels look like this:



Potassium channels **do not have inactivation** gates.

Potassium Activation Variable

Maximum conductivity
with all gates open
($n = 1$)

$$g_K(V, t)$$

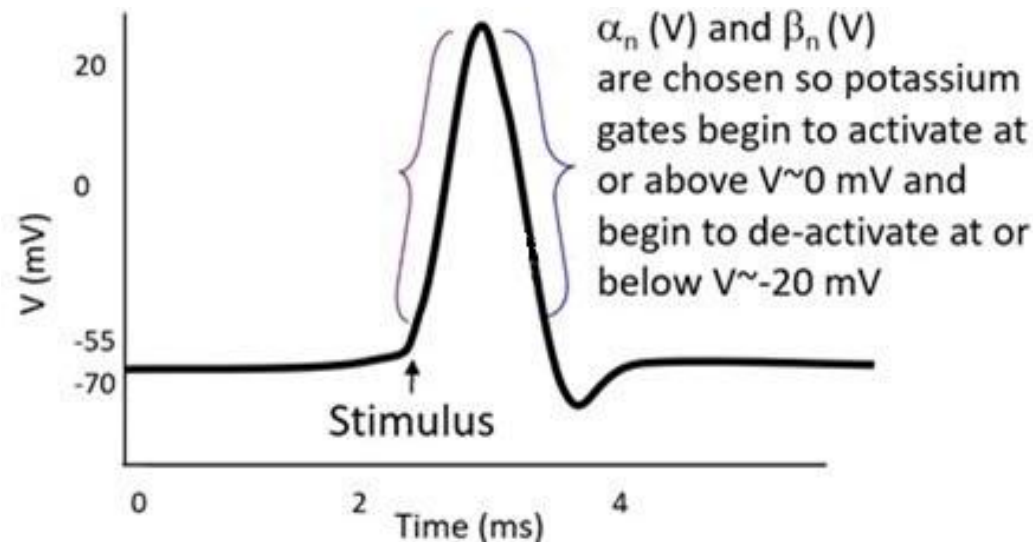
We can write an expression for the conductivity of this ion as

$$g_K = \bar{g}_K n^4(V, t)$$

Four activation gates
result in fourth power
for gating variable n

And then assume a simple first order kinetic behavior of the
gating variable n

$$\frac{dn}{dt} = \alpha_n(V)(1 - n) - \beta_n(V)n \quad (\text{same as previous slide, with } f \text{ now called } n)$$



Ref.: **A Quantitative Description of Membrane Current and its Application to Conduction and Excitation in Nerve**, Hodgkin and Huxley, 1952

Sodium Gating Variables

Inactivation corresponds to value $h=0$, giving $g_{Na} = 0$

$$g_{Na}(V, t)$$

$$g_{Na} = \bar{g}_{Na} m^3(V, t) h(V, t)$$

for which we write a similar ordinary differential equation

$$\frac{dh}{dt} = \alpha_h(V)(1 - h) - \beta_h(V)h$$

One inactivation gate results in first power for gating variable h

The ODE for the activation variable will be:

$$\frac{dm}{dt} = \alpha_m(V)(1 - m) - \beta_m(V)m$$

Sodium Gating Variables

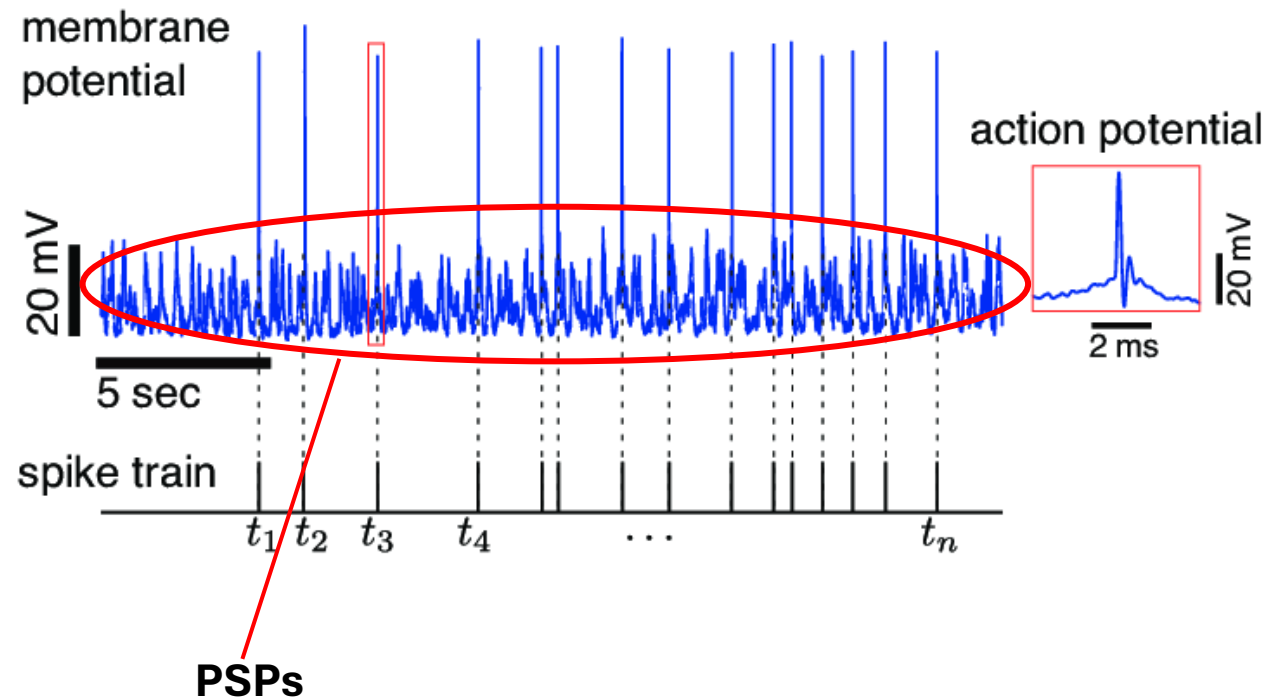
 Ref.: **Sodium channel**, Wikipedia

Action Potential	Membrane Potential	Target Potential	Gate's Target State	Neuron's Target State
Resting	-70 mV	-55 mV	Deactivated → Activated	Polarized
Rising	-55 mV	0 mV	Activated	Polarized → Depolarized
Rising	0 mV	+30 mV	Activated → Inactivated	Depolarized
Falling	+30 mV	0 mV	Inactivated	Depolarized → Repolarized
Falling	0 mV	-70 mV	Inactivated	Repolarized
Undershot	-70 mV	-75 mV	Inactivated → Deactivated	Repolarized → Hyperpolarized
Rebounding	-75 mV	-70 mV	Deactivated	Hyperpolarized → Polarized

$$\begin{aligned}
 C \dot{V} &= I - \overbrace{\bar{g}_K n^4 (V - E_K)}^{I_K} - \overbrace{\bar{g}_{Na} m^3 h (V - E_{Na})}^{I_{Na}} - \overbrace{g_L (V - E_L)}^{I_L} \\
 \dot{n} &= \alpha_n(V)(1 - n) - \beta_n(V)n \\
 \dot{m} &= \alpha_m(V)(1 - m) - \beta_m(V)m \\
 \dot{h} &= \alpha_h(V)(1 - h) - \beta_h(V)h,
 \end{aligned}$$

Spike Train

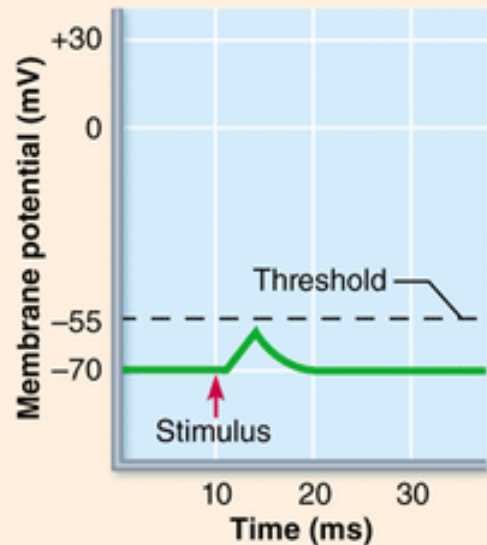
The temporal sequence of spikes generated by a neuron is called “**spike train**”:



Postsynaptic Potential (PSPs)

Synaptic currents produce changes in the **postsynaptic** neurons. These changes are called **postsynaptic potentials** (PSPs).

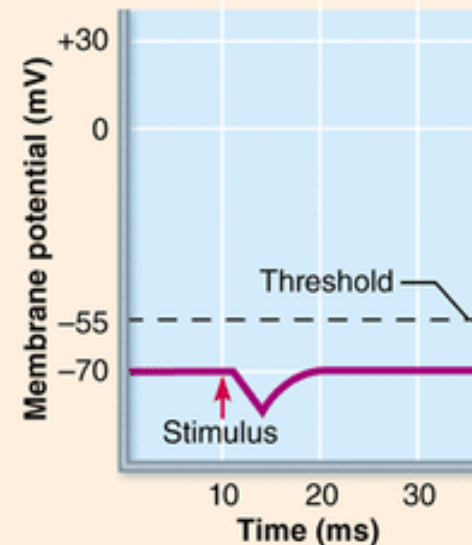
(a) Excitatory postsynaptic potential (EPSP)



An **EPSP** is a local depolarization of the postsynaptic membrane.

- EPSPs bring the neuron closer to AP threshold.
- Neurotransmitter binding opens chemically gated ion channels, allowing Na^+ and K^+ to pass simultaneously.

(b) Inhibitory postsynaptic potential (IPSP)



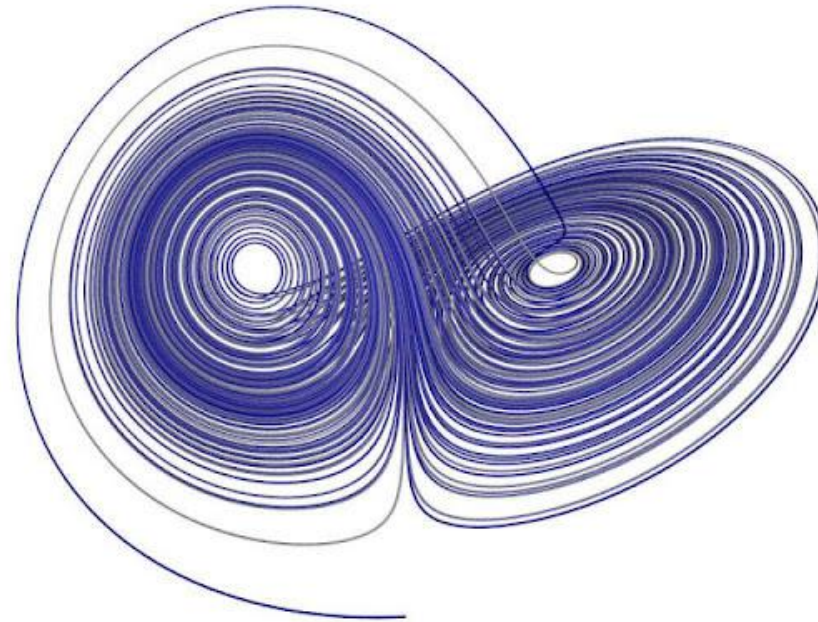
An **IPSP** is a local hyperpolarization of the postsynaptic membrane.

- IPSPs drive the neuron away from AP threshold.
- Neurotransmitter binding opens chemically gated ion channels permeable to either K^+ or Cl^- .

Ref.: **Characterizing Neurotransmitter Receptor Activation with a Perturbation Based Decomposition Method**, Stephen Jue

Neurons as Dynamical Systems

A dynamical system consists of a set of **variables** that describe its **state**, and a law that describes the **evolution** of the state with time.



The State



Dynamical System = states + evolution

The state of **most** models is usually described by the membrane potential V , and the activation (**recovery**) variable n , of the **persistent** K^+ current (2D). The activation (**excitation**) variable of the **transient** Na^+ current is a function of V , so it's not a separate variable.

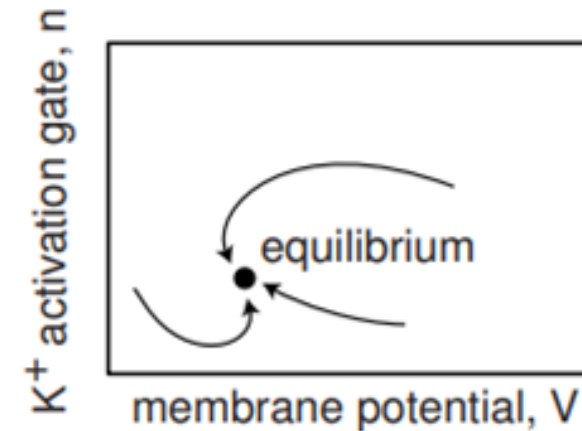
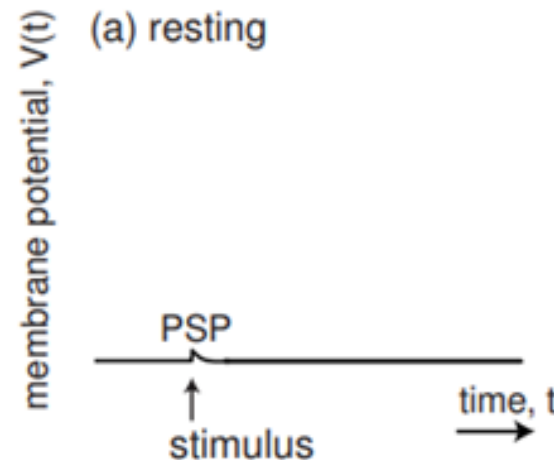
What about the **evolution law**?



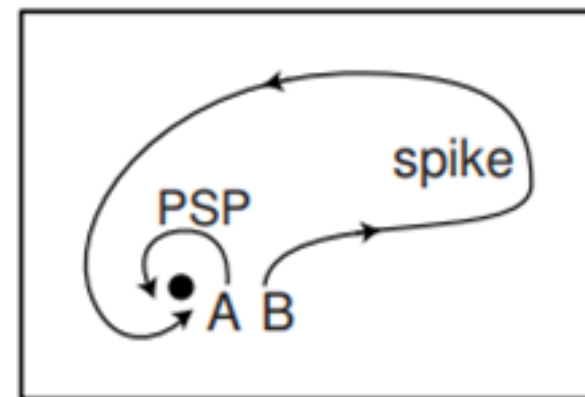
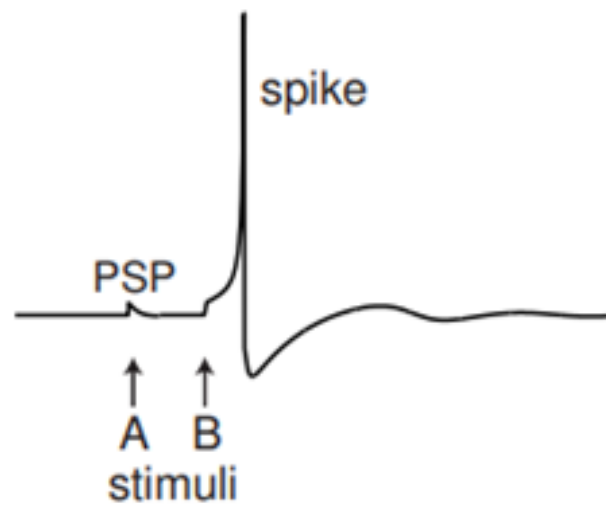
Non-Excitable Phase Portrait

Studying the **phase portrait** of a system, we obtain the qualitative description of the dynamics of that system.

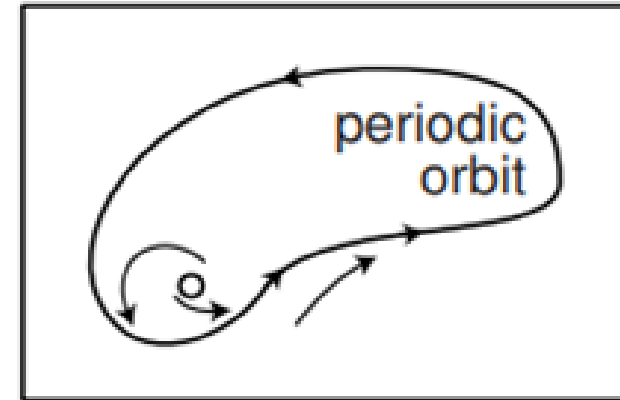
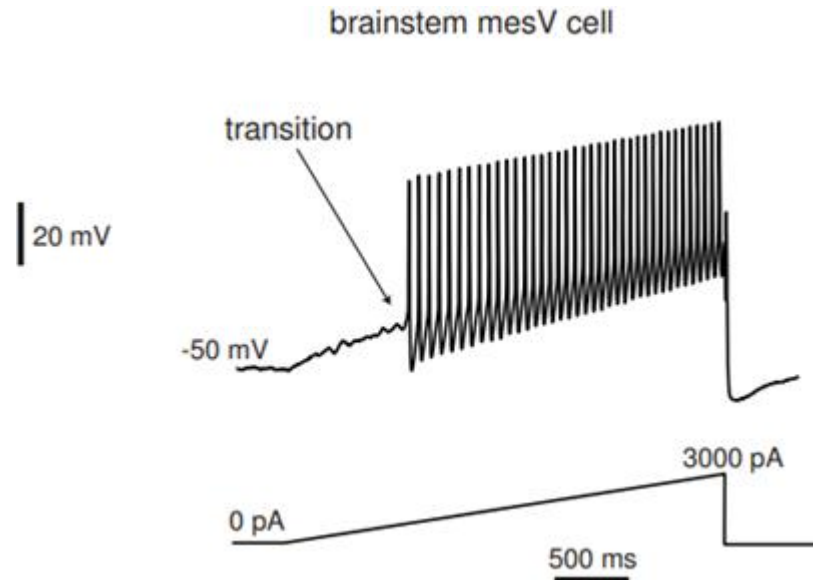
The evolution is a trajectory $(V(t), n(t))$ on the $V \times n$ - plane. The trajectories in the figure are **attracted** to the **stable equilibrium**.



Excitable Phase Portrait



Bifurcations



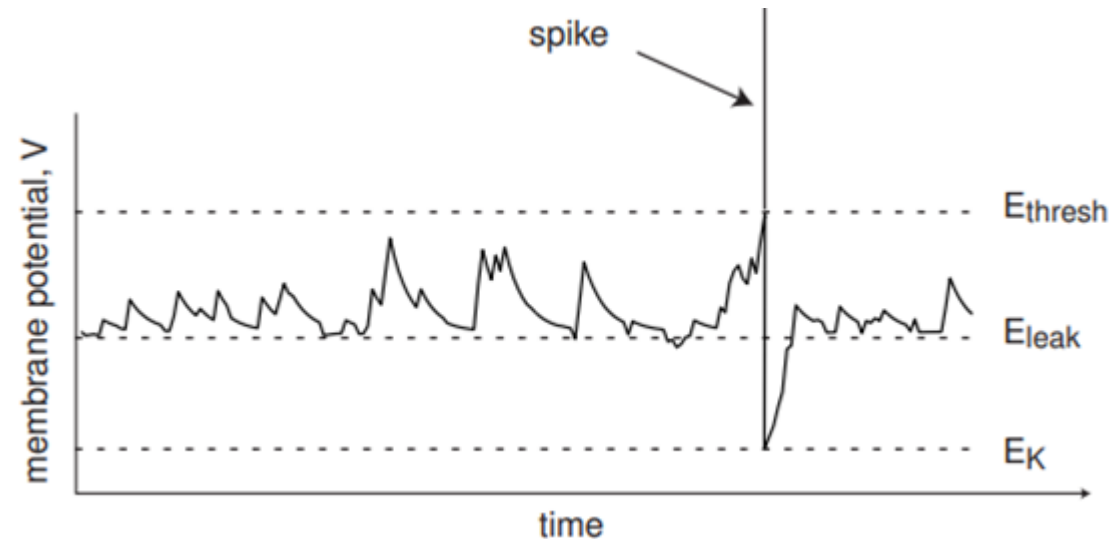
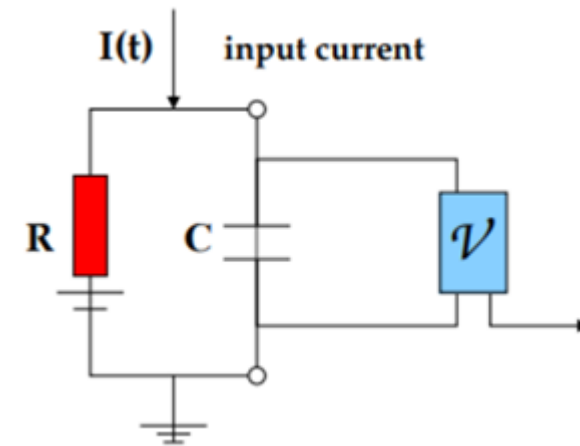
From a dynamical systems POV, the transition from resting to tonic spiking, corresponds to a **bifurcation** of the neuron dynamics, that is, a **qualitative** change of the phase portrait.

Leaky-Integrate-And-Fire (LIF) Model

The leaky integrate-and-fire model (Lapicque, 1907) is an idealization of a neuron having an Ohmic leakage current and voltage-gated currents that are completely deactivated at rest.

The subthreshold behavior can be described by the linear differential equation:

$$C\dot{V} = I - \overbrace{g_{\text{leak}}(V - E_{\text{leak}})}^{\text{Ohmic leakage}},$$

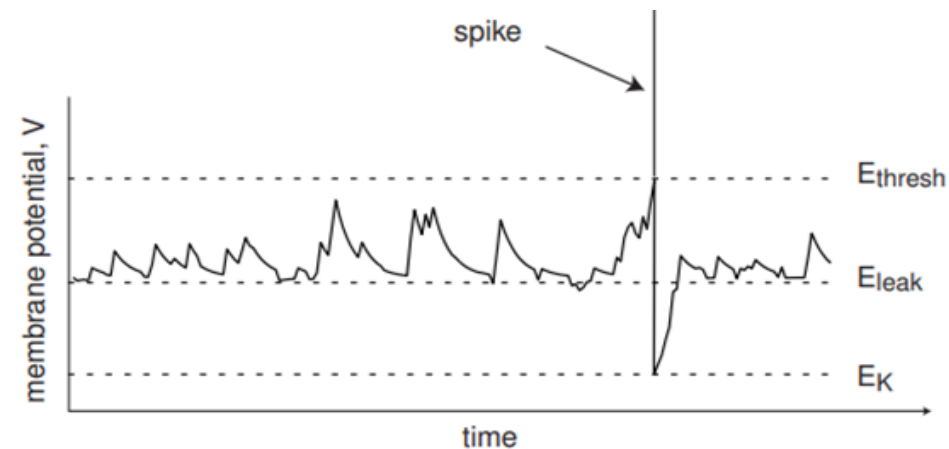


Leaky-Integrate-And-Fire (LIF) Model

After appropriate scaling, the LIF model can be written as:

$$\dot{v} = b - v, \quad \text{if } v = 1, \text{ then } v \leftarrow 0,$$

The resting state is $v = b$, the threshold is $v = 1$, and the reset value is $v = 0$. The neuron is excitable when $b < 1$ and fires periodic spikes when $b > 1$.



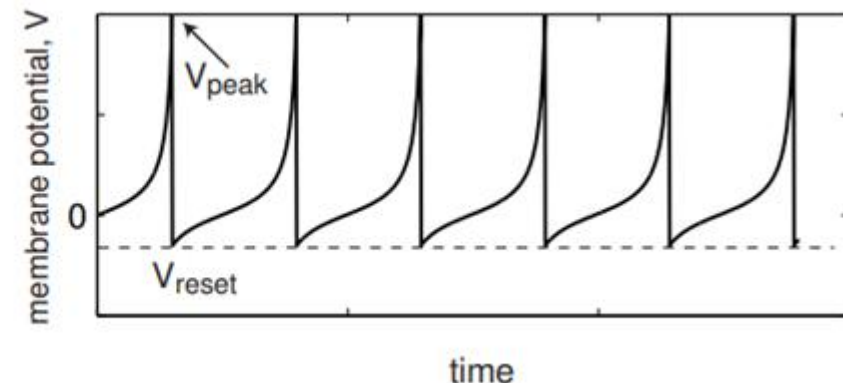
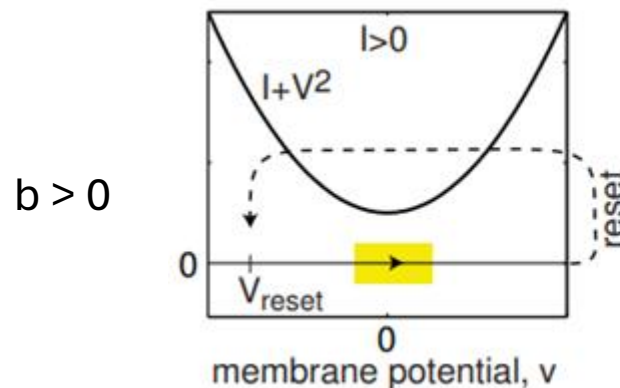
Quadratic-Integrate-And-Fire (QIF) Model

Replacing $-v$, in LIF, with $+v^2$, we obtain the quadratic integrate-and-fire model:

$$\dot{v} = b + v^2, \quad \text{if } v = v_{\text{peak}}, \text{ then } v \leftarrow v_{\text{reset}},$$

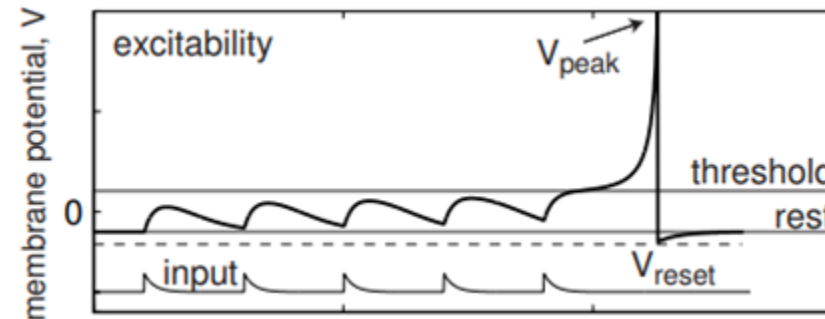
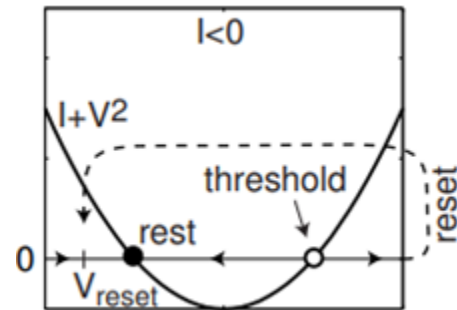
We can normalize $v_{\text{peak}} = 1$
(all-or-none spike)

$b + v^2$ is the topological normal form for the previous bifurcation and resetting v , captures the recurrence we have with tonic spiking.



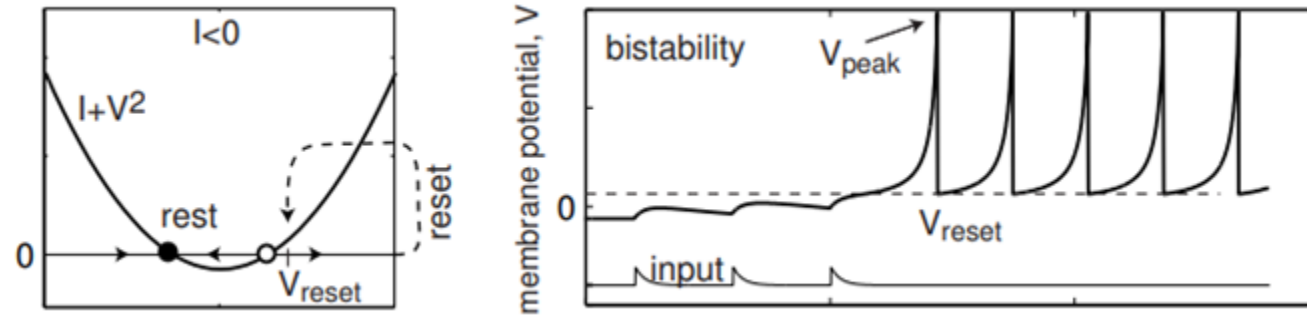
Quadratic-Integrate-And-Fire (QIF) Model

$b < 0$



Quadratic-Integrate-And-Fire (QIF) Model

$$b < 0, \\ v_{\text{reset}} > \sqrt{b}$$



Conclusions on the QIF:

Unlike the LIF, this is a "real" **Integrator**, it has a **dynamic threshold**, and it generates spikes with **latencies**.

What about **Resonators**?

Reduction of Multi-Dimensional Models



Using biophysically accurate Hodgkin-Huxley models is **computationally prohibitive**. Using an integrate-and-fire model is computationally **effective**, but incapable of producing rich dynamics exhibited by real neurons.

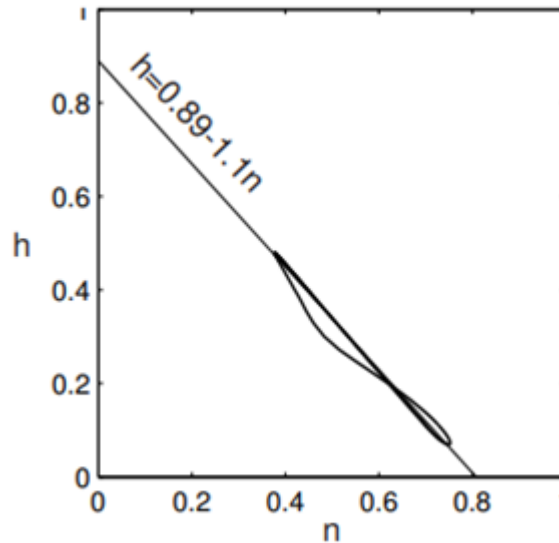
Can we reduce any Hodgkin-Huxley-type model?



Simple 2D Reduction of the HH Model

A simulation, by Krinskii and Kokoz (1973) has shown that there is a relationship between n and h .

One dimension, h , is already gone.



(n, h) - plane

If we also assume that the activation of the Na^+ current is instantaneous ($m = \alpha / (\alpha + \beta)$), then we can remove m .

Resulting 2D Model

$$\begin{aligned}
 C \dot{V} &= I - \overbrace{g_K n^4 (V - E_K)}^{I_K} - \overbrace{g_{Na} m_\infty^3(V) (0.89 - 1.1n) (V - E_{Na})}^{\text{instantaneous } I_{Na}} - \overbrace{g_L (V - E_L)}^{I_L} , \\
 \dot{n} &= (n_\infty(V) - n) / \tau_n(V) ,
 \end{aligned}$$

$$\begin{aligned}
 \dot{n} &= (n_\infty(V) - n) / \tau_n(V) , \\
 \dot{m} &= (m_\infty(V) - m) / \tau_m(V) ,
 \end{aligned}$$

$$\begin{aligned}
 n_\infty &= \alpha_n / (\alpha_n + \beta_n) , & \tau_n &= 1 / (\alpha_n + \beta_n) , \\
 m_\infty &= \alpha_m / (\alpha_m + \beta_m) , & \tau_m &= 1 / (\alpha_m + \beta_m) ,
 \end{aligned}$$

This model's solutions agree quantitatively and qualitatively, to those of the original 4D one.

Simple 2D Reduction - Revamped

In cases involving **large-scale simulations**, the shape of the spike is less important than the subthreshold dynamics. Thus, we can simplify most models into:

$$\begin{aligned}\dot{v} &= I + v^2 - u \\ \dot{u} &= a(bv - u)\end{aligned}$$

if $v \geq 1$, then

$$v \leftarrow c, u \leftarrow u + d$$

?

Depending on the values of **a** and **b**, the neuron can be an **Integrator** or a **Resonator**.

a is the recovery time constant. When **b** ≤ 0 , the model acts as the **QIF** model.

The **sign of b** determines whether **u** is an **amplifying** ($b < 0$) or a **resonant** ($b > 0$) variable, so **Rebound Spikes** are **possible**.

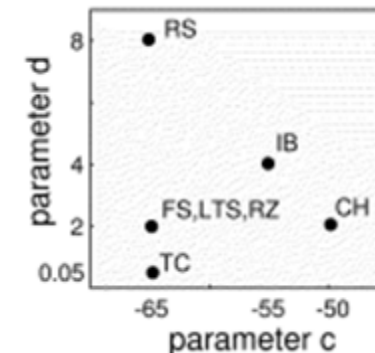
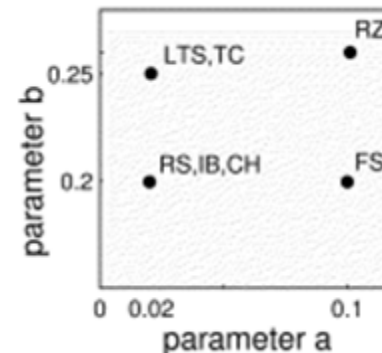
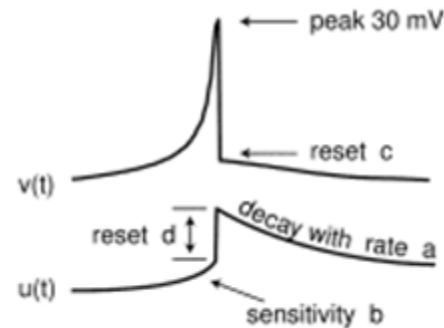
Simulating Neurons & Simulink

The most popular simulation of mammalian neurons is the **Izhikevich's model**, a scaled version of the previous reduction. We are going to show how it reproduces the behaviors of known types of (not only) cortical neurons.

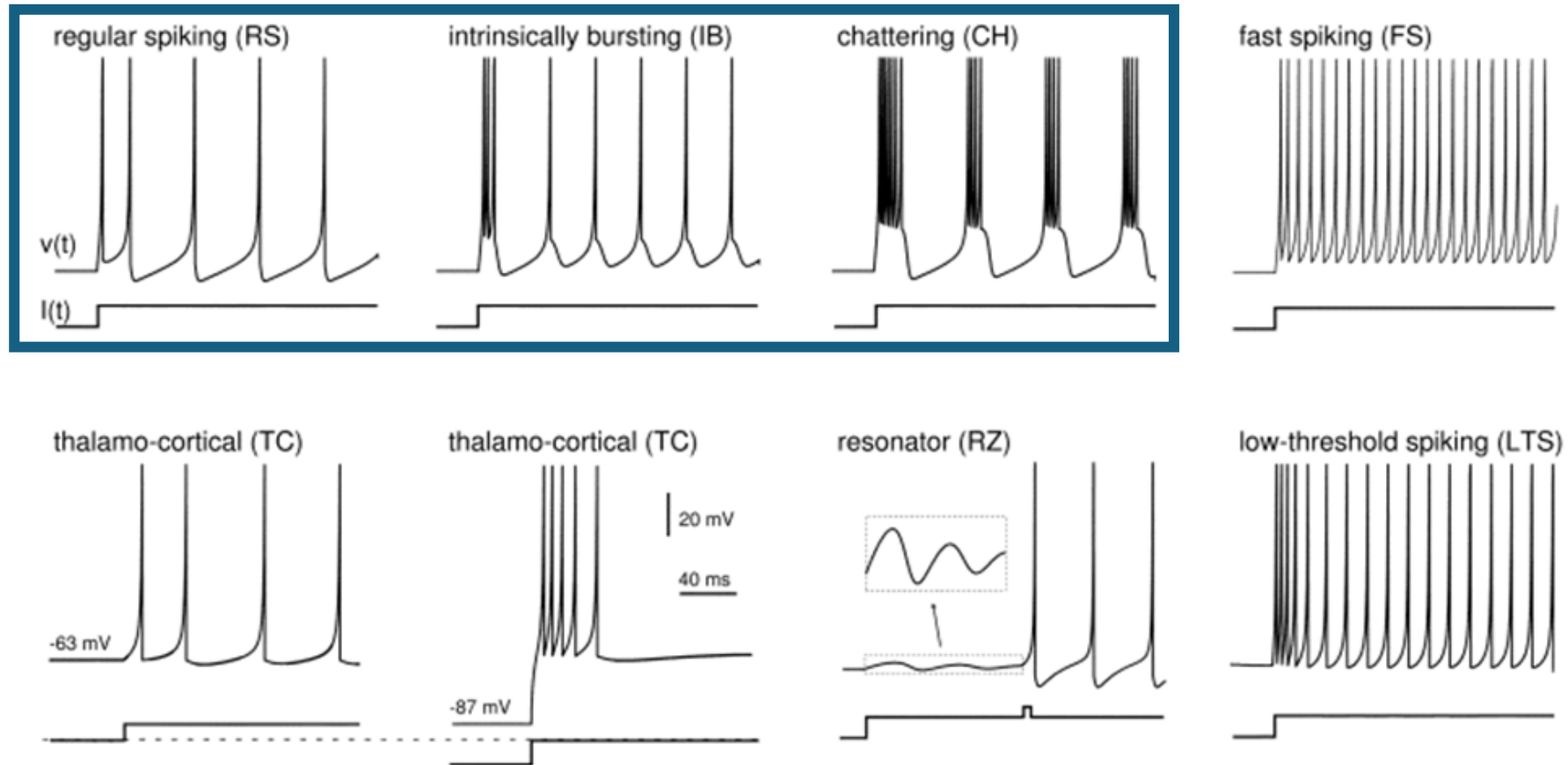
$$v' = 0.04v^2 + 5v + 140 - u + I$$

$$u' = a(bv - u)$$

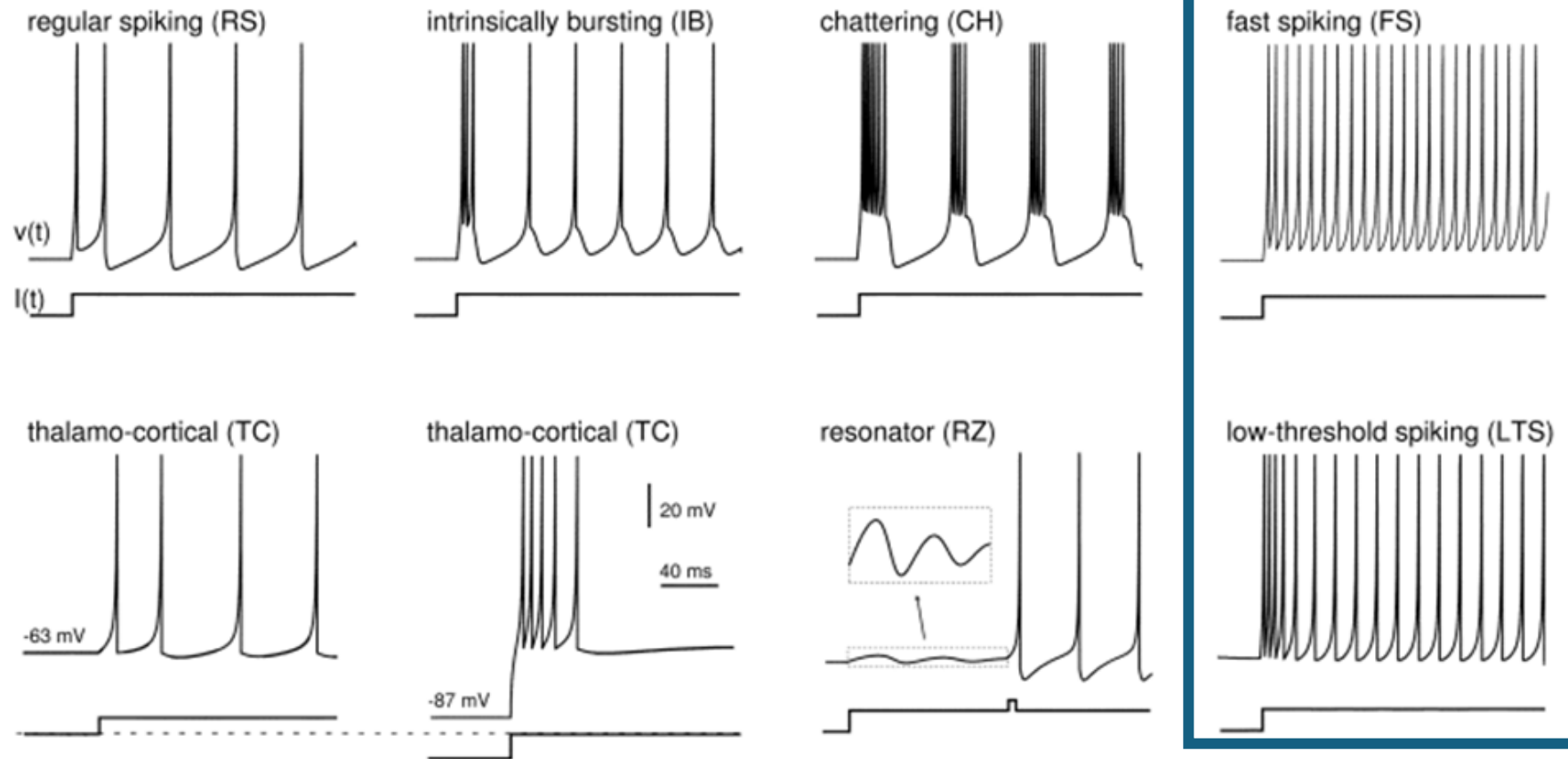
if $v = 30$ mV,
then $v \leftarrow c, u \leftarrow u + d$



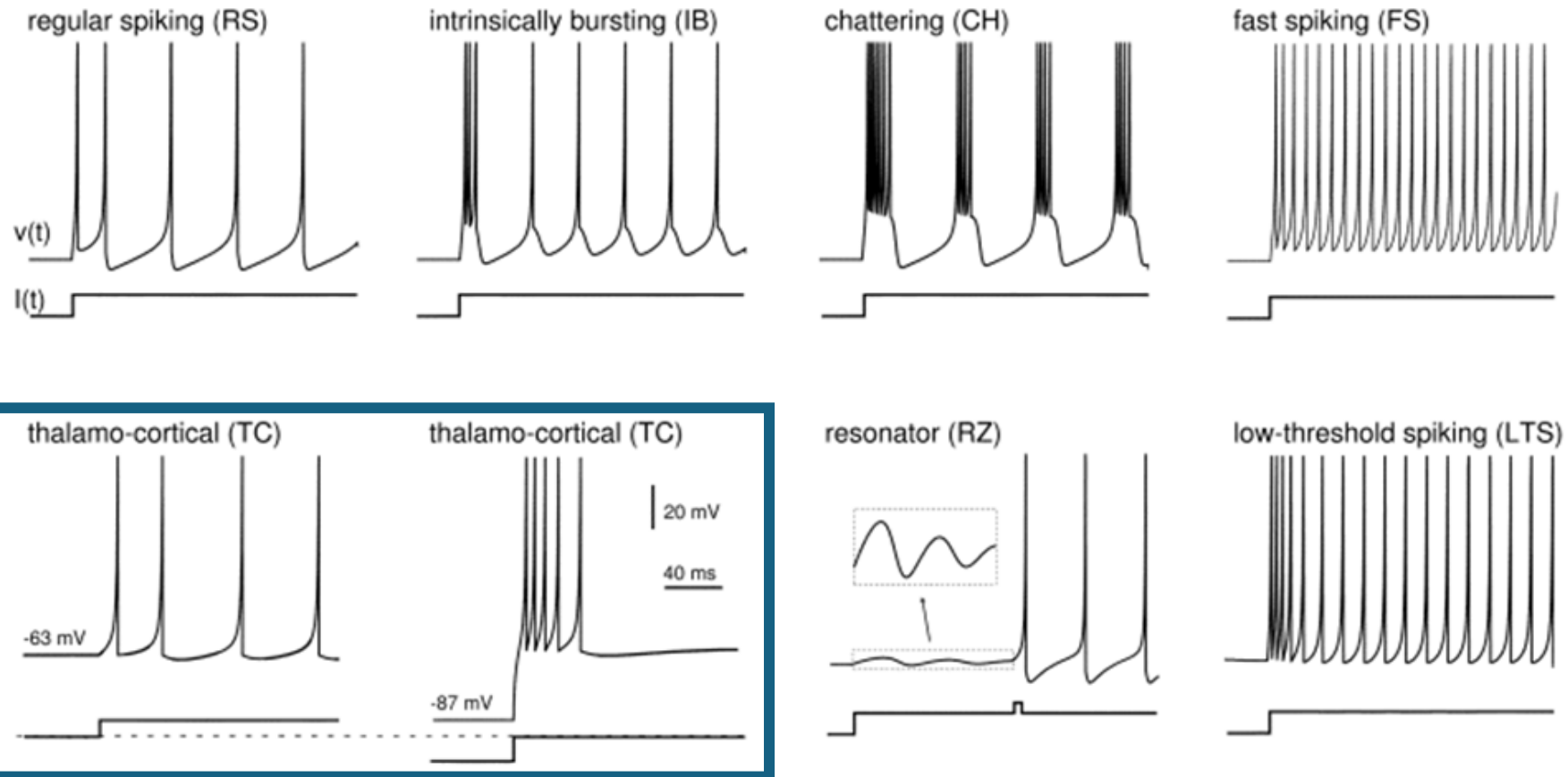
Izhikevich's Model: Cortical Excitatory Neurons



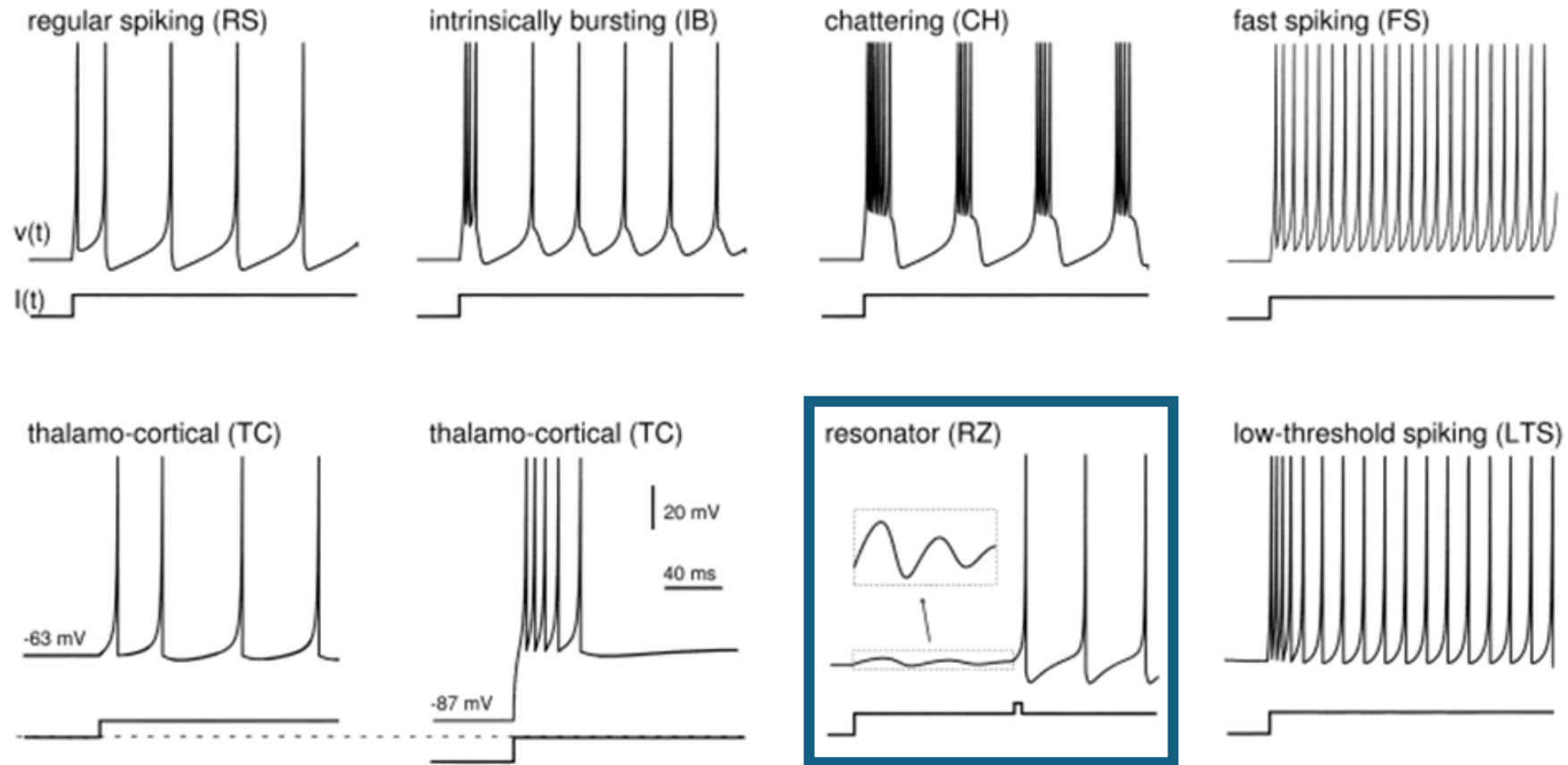
Izhikevich's Model: Inhibitory Cortical Neurons



Izhikevich's Model: Thalamo-Cortical Neurons



Izhikevich's Model: Resonators



Izhikevich's Model: Resonator Code

```
# Liste per salvare i risultati
v_trace = []
u_trace = []
```

```
# Parametri del modello di Izhikevich per il neurone Resonator
a = 0.1
b = 0.26
c = -60
d = -1

# Condizioni iniziali
v = -62 # Potenziale di membrana iniziale (mV)
u = b * v # Valore iniziale di u

# Simulazione
tau = 0.25 # Passo temporale (ms)
time = np.arange(0, 400, tau) # Tempo totale di simulazione (ms)
I = np.zeros(len(time)) # Corrente di ingresso

# Definizione degli impulsi di corrente
T1 = time[-1] / 10
T2 = T1 + 20
T3 = 0.7 * time[-1]
T4 = T3 + 40

for i, t in enumerate(time):
    if (T1 < t < T1 + 4) or (T2 < t < T2 + 4) or (T3 < t < T3 + 4) or (T4 < t < T4 + 4):
        I[i] = 0.65
```



Izhikevich's Model: Code

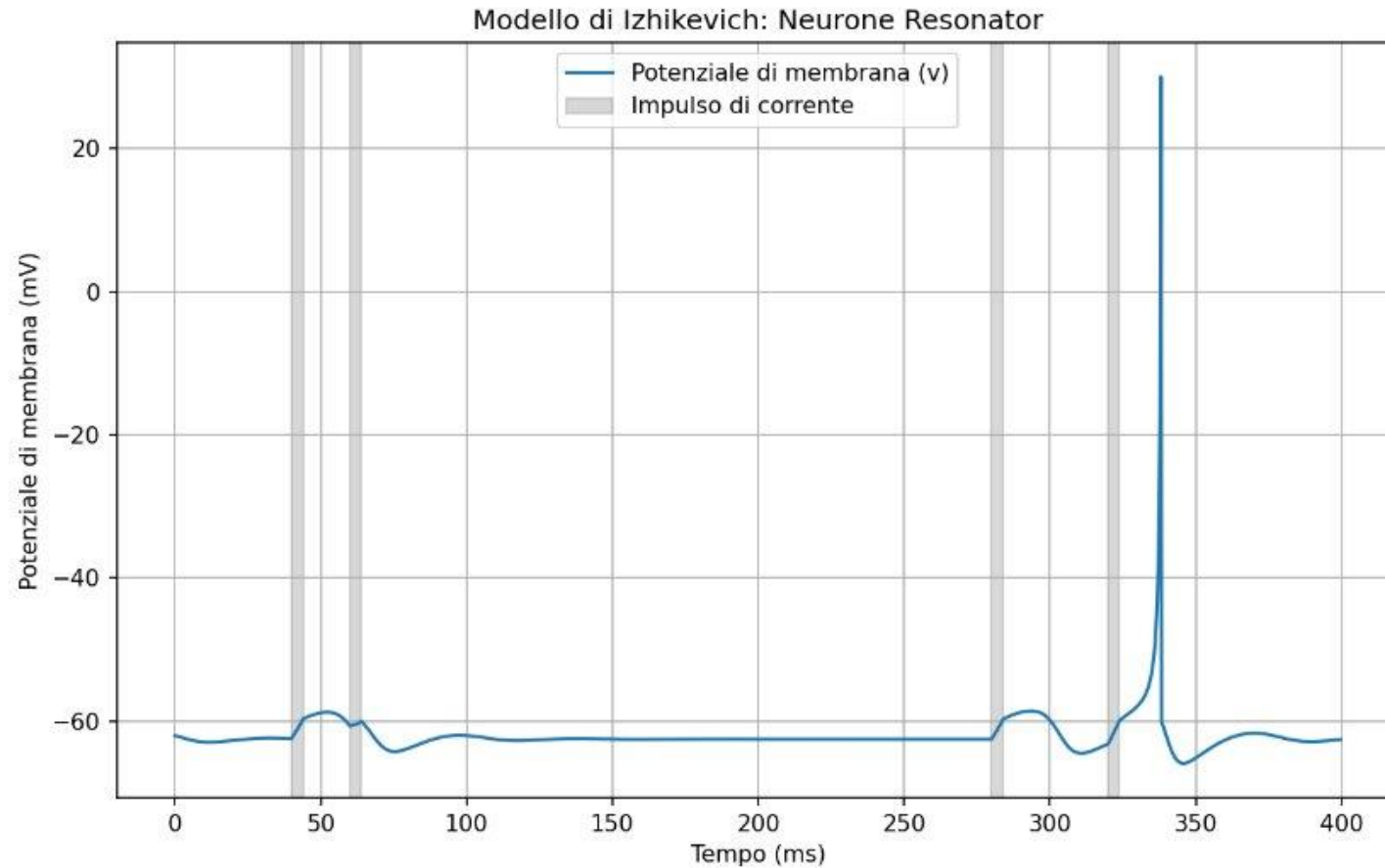
```
for i, t in enumerate(time):  
    v_trace.append(v)  
    u_trace.append(u)  
  
    # Equazioni differenziali  
    v = v + tau * (0.04 * v**2 + 5 * v + 140 - u + I[i])  
    u = u + tau * a * (b * v - u)  
  
    # Reset del potenziale e del valore di u  
    if v > 30:  
        v_trace[-1] = 30 # Salva il picco  
        v = c  
        u += d
```

Euler's Explicit Method:

$$v(t + \tau) = v(t) + \tau \cdot \frac{dv}{dt}$$

$$u(t + \tau) = u(t) + \tau \cdot \frac{du}{dt}$$

Izhikevich's Model: Results Plot



Pulse-Coupled Neural Networks (PCNN):

```

Ne = 800
Ni = 200 4:1
re = np.random.rand(Ne)
ri = np.random.rand(Ni)  Heterogeneity
a = np.r_[0.02*np.ones(Ne), 0.02+0.08*ri]
b = np.r_[0.2*np.ones(Ne), 0.25-0.05*ri]
c = np.r_[-65+15*re**2, -65*np.ones(Ni)]
d = np.r_[8-6*re**2, 2*np.ones(Ni)]
S = np.c_[0.5*np.random.rand(Ne+Ni, Ne), -np.random.rand(Ne+Ni, Ni)]

v = -65*np.ones(Ne+Ni)  #Initial values of v.
u = b*v  #Initial values of u.
firings = np.zeros((0,2))

for t in range(1000):  #Stimulation of 1000 ms
    I = np.r_[5*np.random.randn(Ne), 2*np.random.randn(Ni)] #Thalamic input
    fired = np.where(v >= 30)[0] # Indices of spikes
    if len(fired) != 0:
        firings = np.vstack((firings, np.c_[t+0*fired, fired]))
        v[fired] = c[fired]
        u[fired] = u[fired] + d[fired]
        I = I + S[:, fired].sum(1)
        v = v+0.5*(0.04*v**2+5*v+140-u+I)
        v = v+0.5*(0.04*v**2+5*v+140-u+I)
        u = u+a*(b*v-u)

plot.plot(firings[:, 0], firings[:, 1], ".")
plot.title("Izhikevich's simple neuron network model")
plot.show()

```

PCNN

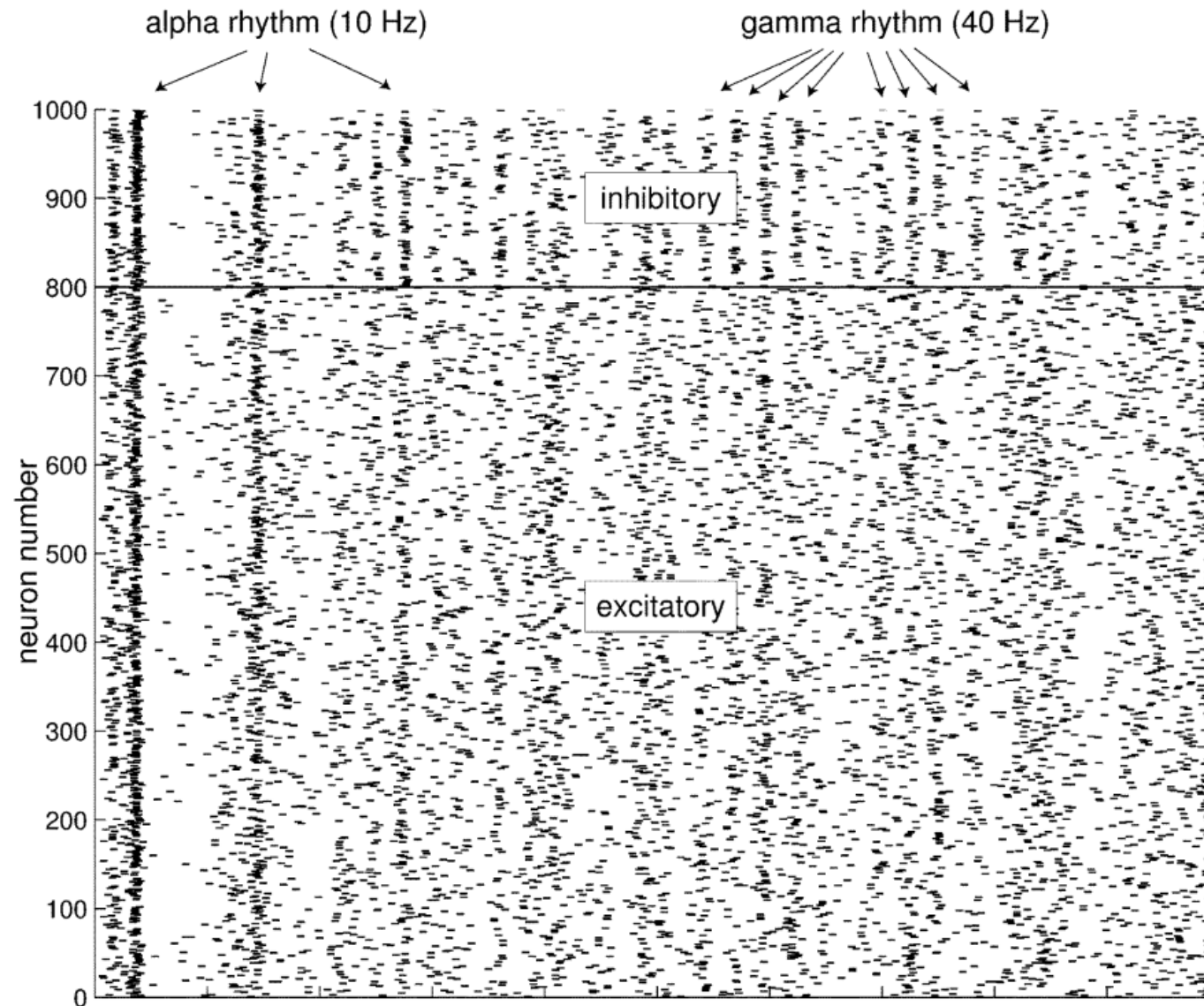
 $S = (s_{ij})$ If "j" fires, $v_i += s_{ij}$ Izhikevich's
EquationsLet i be the neuron index:

excitatory cell $(a_i, b_i) = (0.02, 0.2)$
 and $(c_i, d_i) = (-65, 8) + (15, -6)*re_i^2$

Where re_i , and ri_j , are $\in [0, 1]$,
 uniformly distributed.

Similarly, each inhibitory cell has
 $(a_i, b_i) = (0.02, 0.25) + (0.08, -0.05)*ri_i$ and $(c_i, d_i) = (-65, 2)$.

Pulse-Coupled Neural Networks (PCNN):

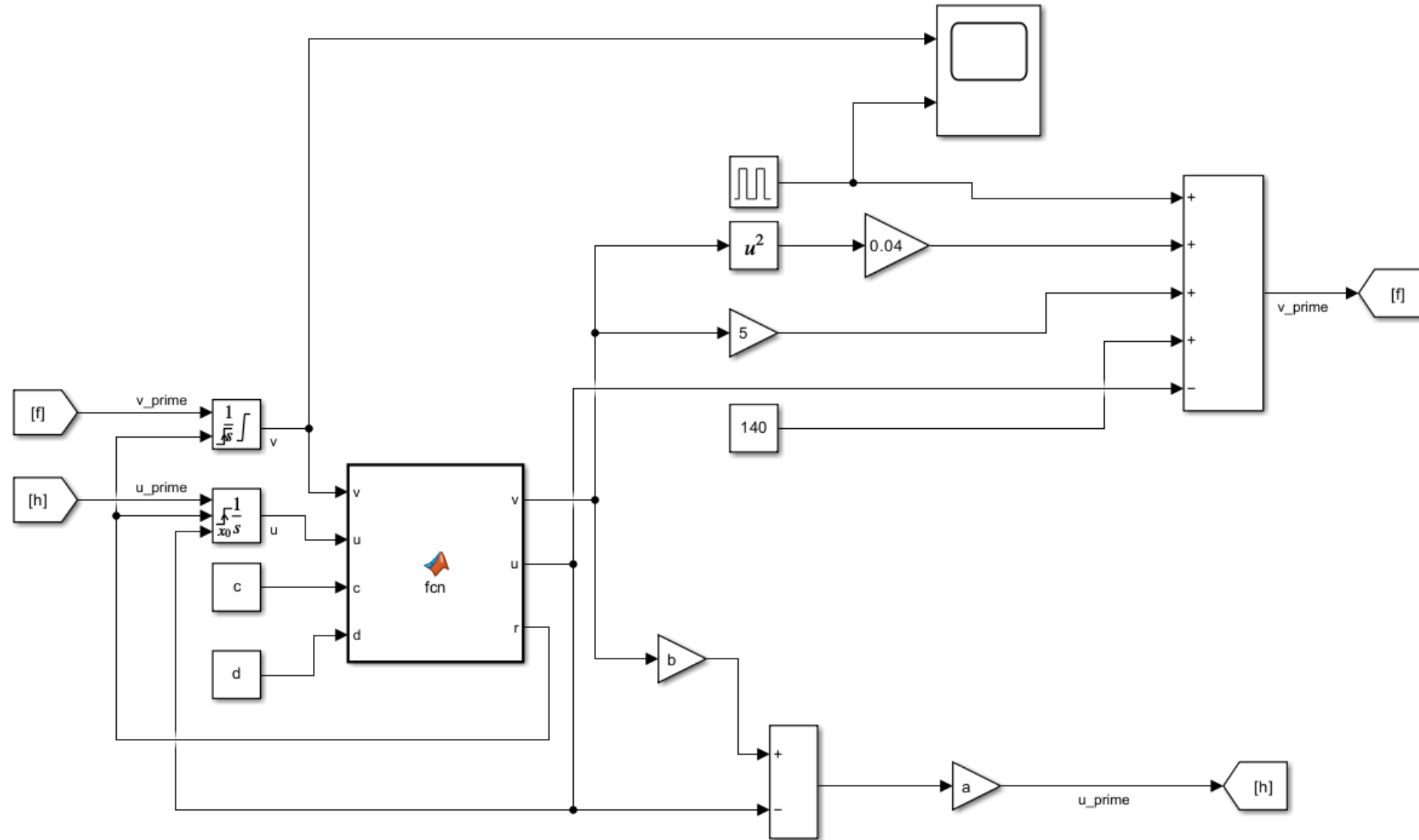


Simulink Implementation:

$$v' = 0.04v^2 + 5v + 140 - u + I$$

$$u' = a(bv - u)$$

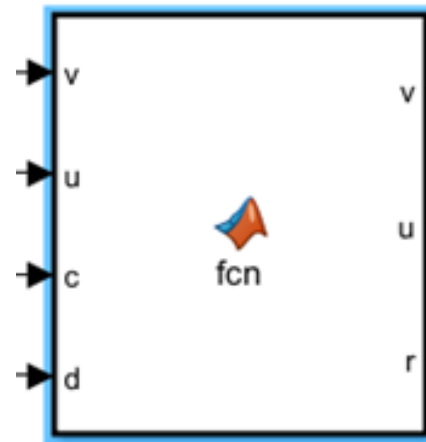
if $v = 30$ mV,
then $v \leftarrow c, \quad u \leftarrow u + d$



Simulink Implementation:

$v' = 0.04v^2 + 5v + 140 - u + I$
 $u' = a(bv - u)$

if $v = 30$ mV,
then $v = c$, $u = u + d$

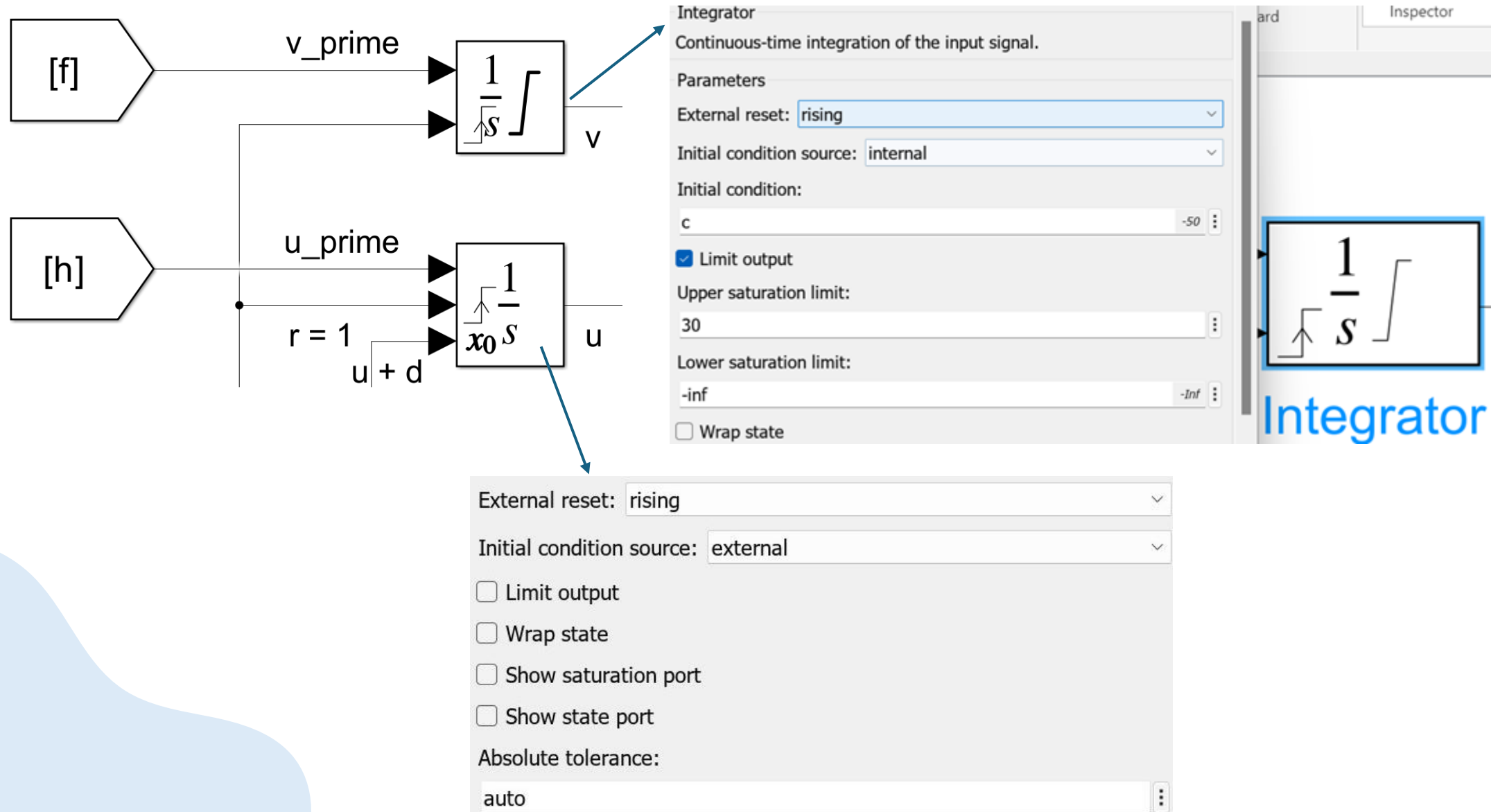


MATLAB Function

```

function [v, u, r] = fcn(v, u, c, d)

if v >= 30
    v = c;
    u = u + d;
    r = 1;
else
    r = 0;
end
  
```

Simulink Implementation:

```
%% Parameter setting
```

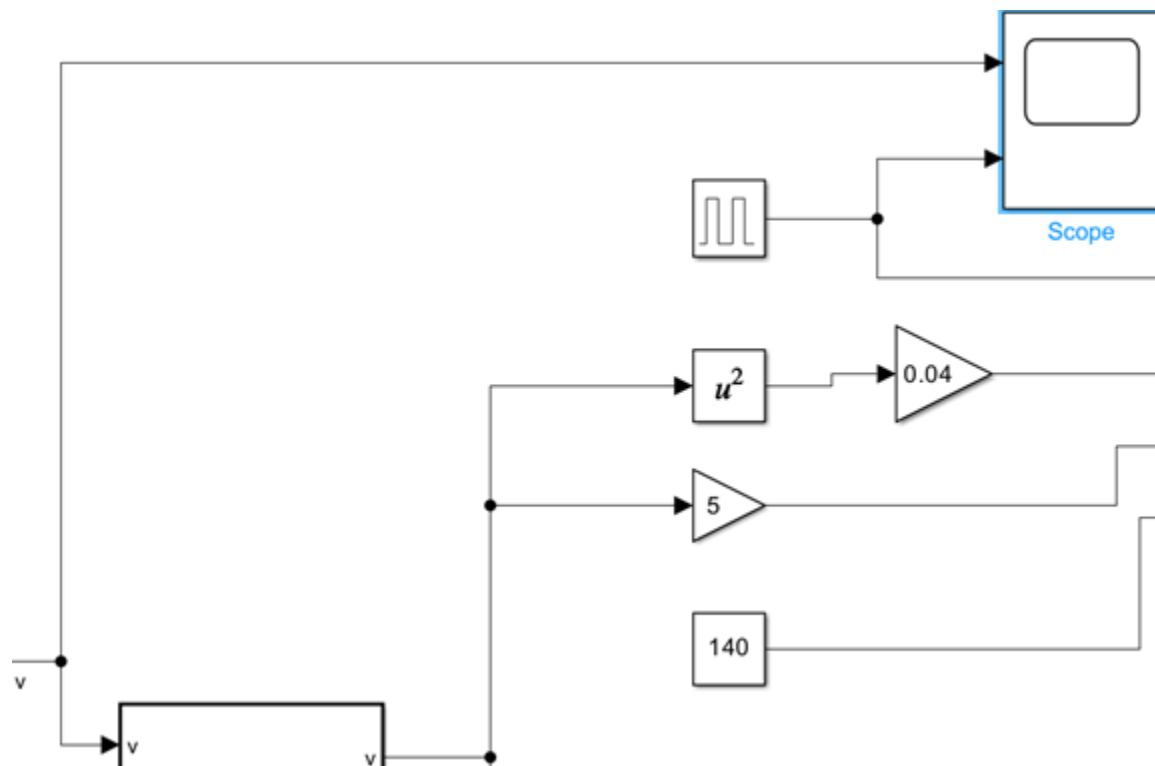
```
pars=[0.02    0.2    -65    6    14    ;...  tonic spiking  
      0.02    0.2    -50    2    15    ]; % tonic bursting
```

```
T = 2;           % select the neuron behaviour  
par = pars(T,:); % parameters for the model
```

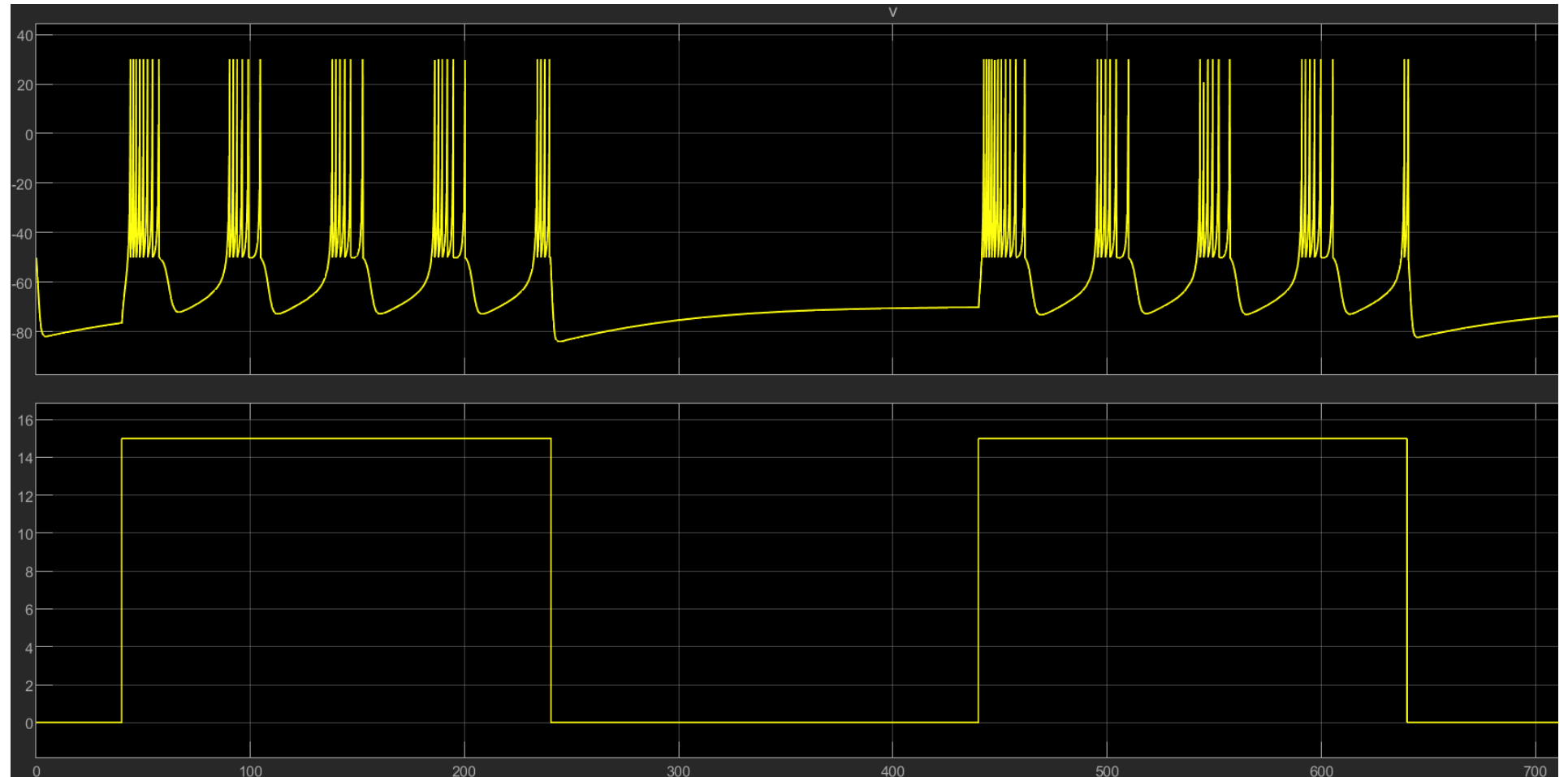
```
a = par(1);  
b = par(2);  
c = par(3);  
d = par(4);  
I = par(5);      % Input current peak
```

```
STOP = 500;      % Simulation time [s]
```

Simulink Implementation:



Simulink Implementation:



EXTRA
SLIDES:



The Importance of Ions:

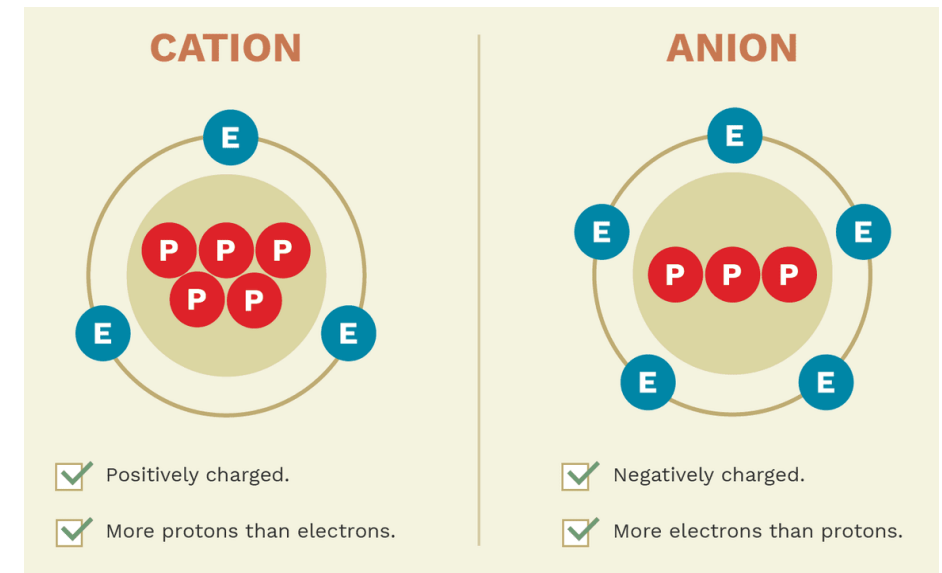
Every neuron maintains a **voltage gradient** across its membrane, due to the differences in ions of **Sodium (Na^+)**, **Potassium (K^+)**, **Chloride (Cl^-)** and **Calcium (Ca^{2+})** in the cell. If the voltage changes significantly, an **electro-chemical** pulse, the **spike**, is fired.

Monovalent Cations

Monovalent Anion

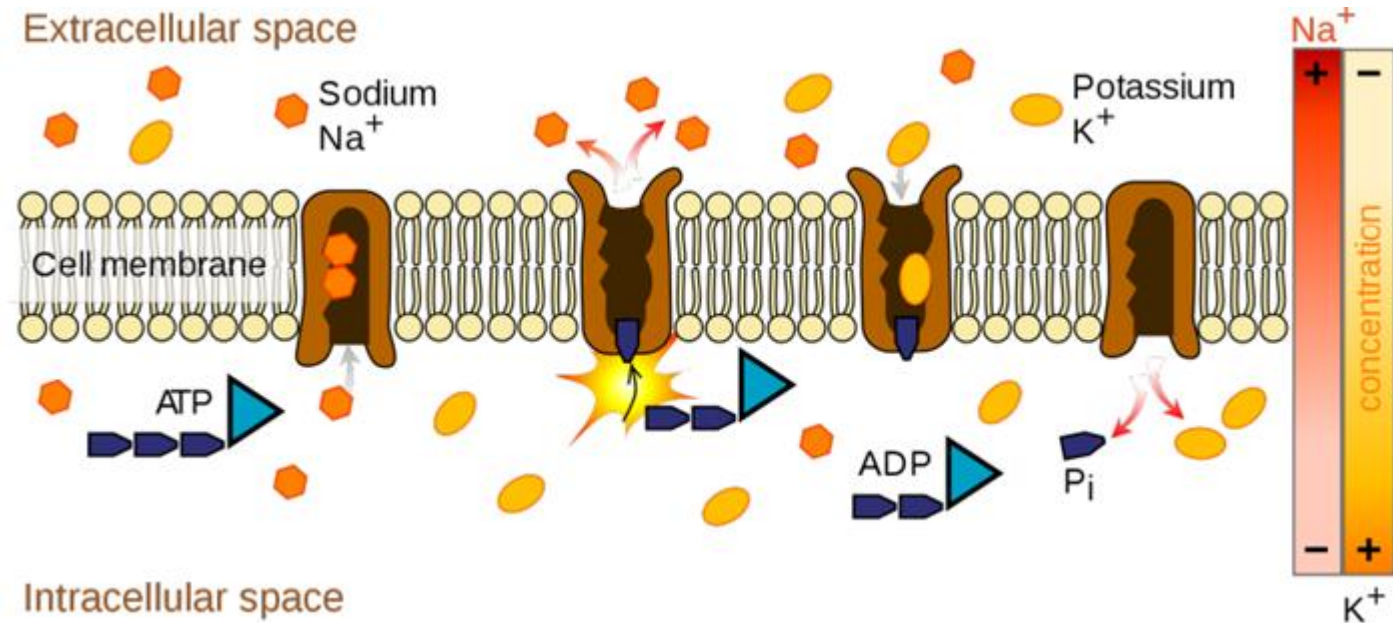
Divalent Cation

capable of bonding with a single hydrogen atom or another element equivalent to it.



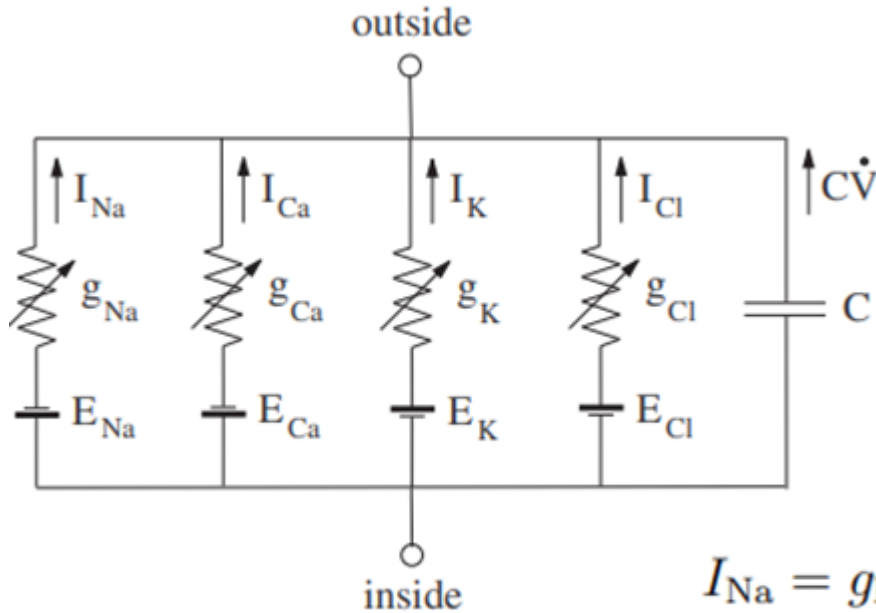
The Sodium-Potassium Pump

Ion pumps maintain the gradient, but spikes involve mainly the use of ion channels. If the ion pumps are turned off, the axon can still fire 100k spikes before their amplitudes begin to decay.



We also have the sodium-calcium exchanger pump, which counteracts the sodium-potassium one.

Equivalent Circuit



Let V be the **membrane potential** and E_{Na} , E_{Ca} , E_K , and E_{Cl} the **Nernst Potentials**. If $V = E_K$, then the net K^+ current, I_K ($\mu A/cm^2$), is zero.

$$I_K = g_K (V - E_K) ,$$

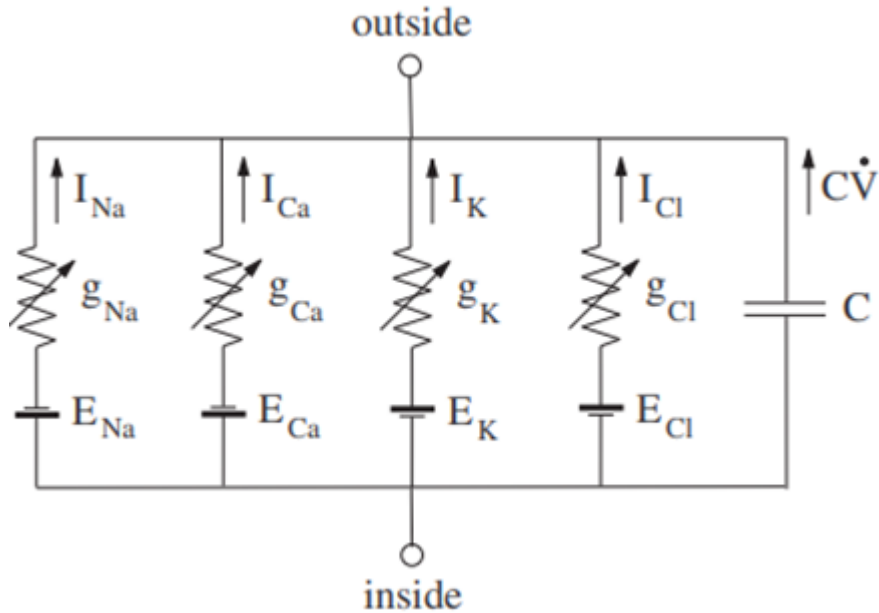
Similarly we can write:

$$I_{Na} = g_{Na} (V - E_{Na}) , \quad I_{Ca} = g_{Ca} (V - E_{Ca}) , \quad I_{Cl} = g_{Cl} (V - E_{Cl}) ,$$

And according to
Kirchhoff's law:

$$I = C\dot{V} + I_{Na} + I_{Ca} + I_K + I_{Cl} ,$$

Equivalent Circuits



But we could also write:

$$C \dot{V} = I - I_{Na} - I_{Ca} - I_K - I_{Cl}$$

Or equivalently

$$C \dot{V} = I - g_{Na} (V - E_{Na}) - g_{Ca} (V - E_{Ca}) - g_K (V - E_K) - g_{Cl} (V - E_{Cl}) .$$

If there are no additional sources, like a synaptic or an injected current, then $I = 0$. This is the **resting state**, the **resting potential** is bounded by the **equilibrium** potentials:

$$E_K < E_{Cl} < V_{(at\ rest)} < E_{Na} < E_{Ca} ,$$

Common Values For HH Model

The capacitance is usually $C \approx 1.0 \mu\text{F}/\text{cm}^2$ in the squid axon

Typical Maximum Conductances:

$$\bar{g}_K = 36 \text{ mS}/\text{cm}^2, \quad \bar{g}_{Na} = 120 \text{ mS}/\text{cm}^2, \quad g_L = 0.3 \text{ mS}/\text{cm}^2.$$

Typical Alpha & Beta Values:

$$\alpha_n(V) = 0.01 \frac{10 - V}{\exp\left(\frac{10 - V}{10}\right) - 1},$$

$$\beta_n(V) = 0.125 \exp\left(\frac{-V}{80}\right),$$

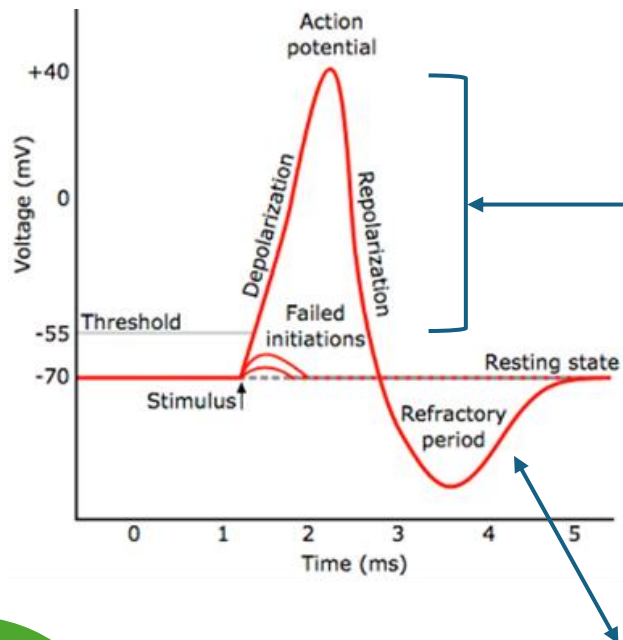
$$\alpha_m(V) = 0.1 \frac{25 - V}{\exp\left(\frac{25 - V}{10}\right) - 1},$$

$$\beta_m(V) = 4 \exp\left(\frac{-V}{18}\right),$$

$$\alpha_h(V) = 0.07 \exp\left(\frac{-V}{20}\right),$$

$$\beta_h(V) = \frac{1}{\exp\left(\frac{30 - V}{10}\right) + 1}.$$

Summarizing



After a spike is fired, there is a negative shift, called **after-hyperpolarization** or **undershoot**.

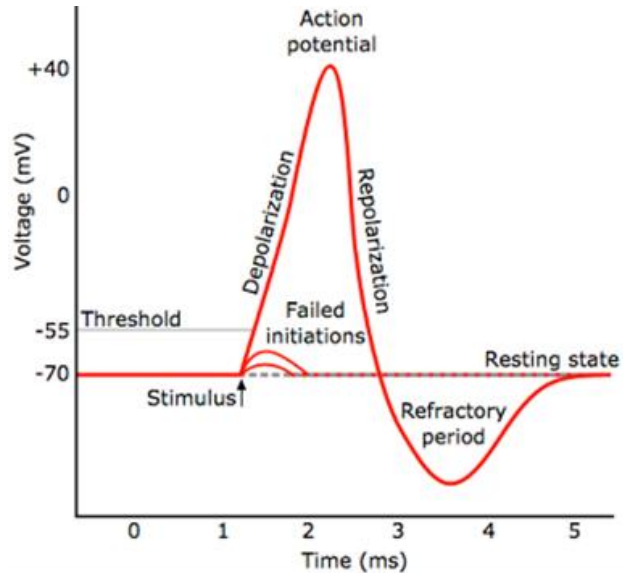
Voltage-Gated **Sodium** channels **rapidly open** when the potential increases to the **threshold** voltage, depolarizing the membrane.

This causes more channels to open, until they are all open, causing the polarity of the membrane to **reverse**.

Then sodium channels **inactivate**, in turn, potassium channels activate. The outward current of potassium helps resetting the gradient to the resting state.

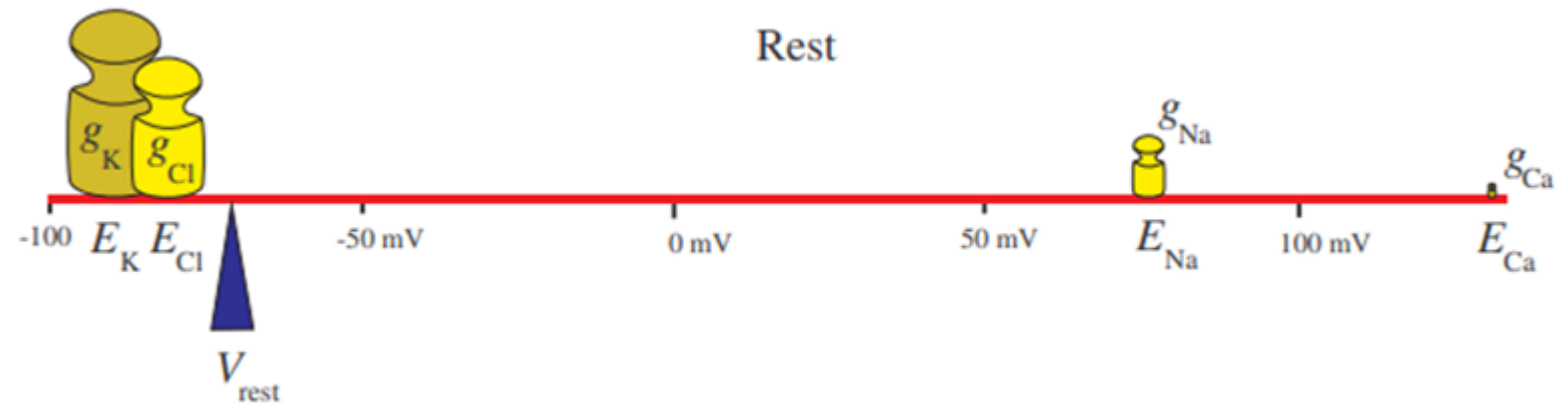
Threshold \in
[-55, -40] mV

The Resting Potential



The potential of quiescent cells is called “**resting potential**”. It’s a stable value at which all inward and outward currents balance each other so that the net current is zero ($I = 0$).

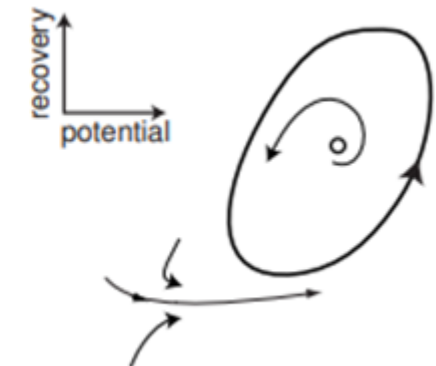
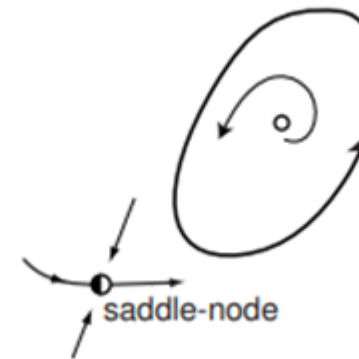
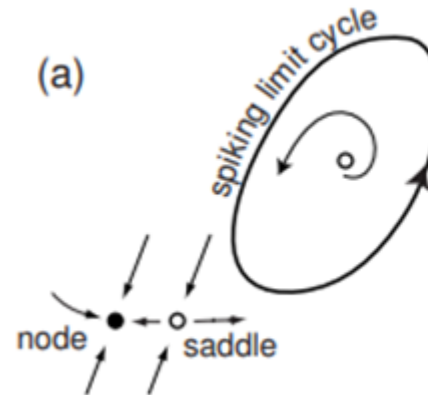
$$V_{\text{rest}} = \frac{g_{\text{Na}}E_{\text{Na}} + g_{\text{Ca}}E_{\text{Ca}} + g_{\text{K}}E_{\text{K}} + g_{\text{Cl}}E_{\text{Cl}}}{g_{\text{Na}} + g_{\text{Ca}} + g_{\text{K}} + g_{\text{Cl}}} \approx 70\text{mV}$$



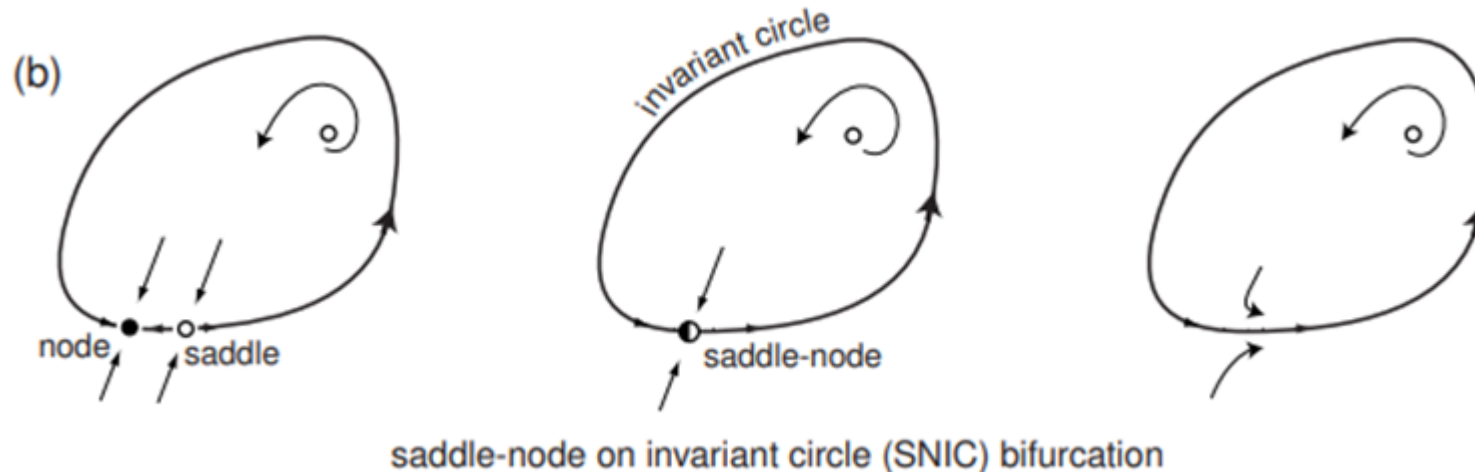
Types of Bifurcations

The type of bifurcation depends on the neuron's physiology and determines its excitable properties. There are four major types of bifurcations: Saddle Node, Saddle-Node on Invariant Circle, Subcritical Andronov-Hopf Bifurcation, Supercritical Andronov-Hopf Bifurcation

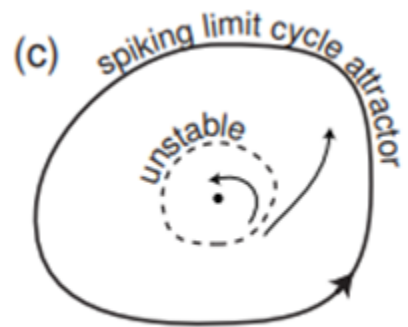
Saddle Node Bifurcation:



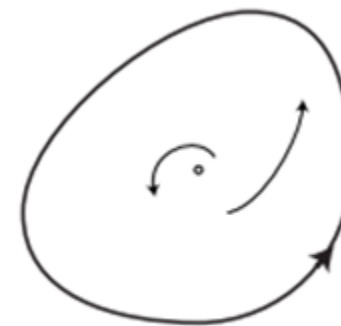
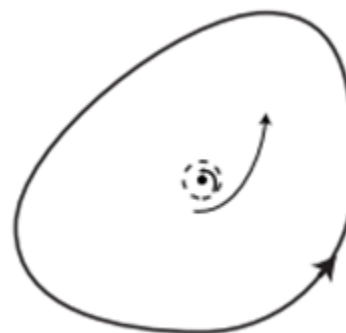
Types of Bifurcations



Types of Bifurcations



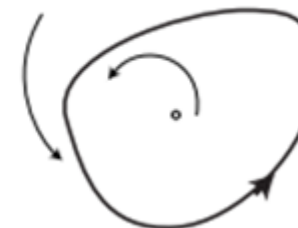
subcritical Andronov-Hopf bifurcation



(d)



supercritical Andronov-Hopf bifurcation



Neurons Classification

		coexistence of resting and spiking states	
		YES (bistable)	NO (monostable)
subthreshold oscillations	NO (integrator)	saddle-node	saddle-node on invariant circle
	YES (resonator)	subcritical Andronov-Hopf	supercritical Andronov-Hopf

Izhikevich's Own Interactive Tool:

