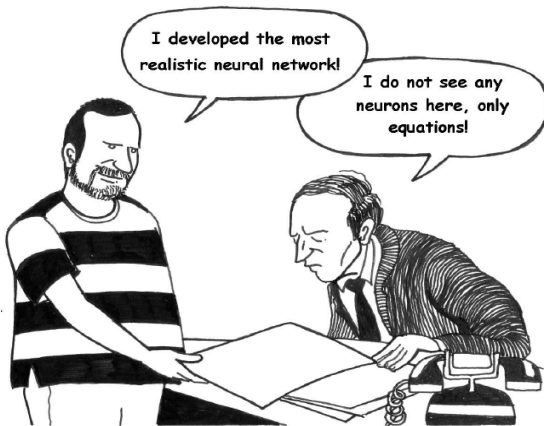


Lecture 2: Mathematical modelling of biological neurons

- To derive the Hodgkin-Huxley neuron model, obtained from experiments on a squid giant axon, and which provides us with the most biophysically realistic equations for describing neuronal dynamics.



- The neuron model was developed by **Alan L. Hodgkin and Andrew F. Huxley in 1952** using the giant squid axon, earning them a **Nobel Prize in Physiology or Medicine 1963**. This approach was refined over the next 50 years and is now known as the **conductance-based approach to modeling neurons**.

The Nobel Prize in Physiology or Medicine 1963



Photo from the Nobel Foundation archive.

Sir John Carew Eccles

Prize share: 1/3

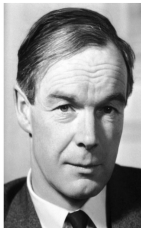


Photo from the Nobel Foundation archive.

Alan Lloyd Hodgkin

Prize share: 1/3

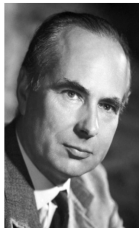
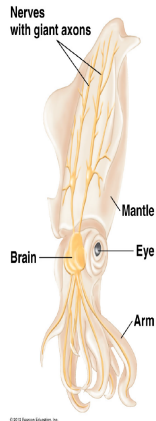


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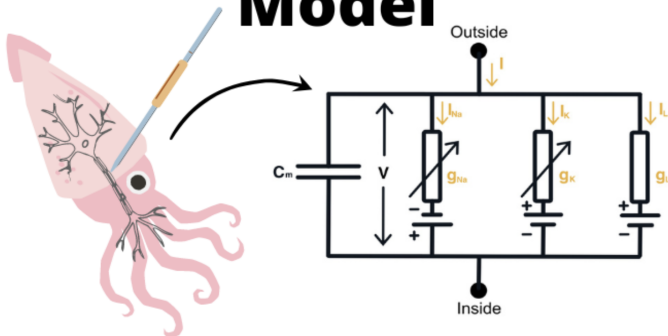
Andrew Fielding Huxley

Prize share: 1/3



- The model simplifies the electrochemical mechanism by representing the neuron membrane as a **capacitor**, **ionic gates**, and **ionic pumps** as conductance-battery pairs. The figure below illustrates the electrical circuit.

Hodgkin-Huxley Model



Circuit diagram for Hodgkin-Huxley (HH) equations.

- Hodgkin and Huxley found that there are **three** main types of currents describing the dynamics of a neuron: Sodium (Na^+), Potassium (K^+), and a leak current (consisting of Chloride (Cl^-) ions).

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- There are biological constructs in a neuron's membrane that gate the flow of specific ions depending on the membrane potential V .
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- There are biological constructs in a neuron's membrane that gate the flow of specific ions depending on the membrane potential V .
- Voltage-gated ion channels control the flow of these ions. When the neuron's membrane reaches a specific voltage, it opens up, allowing these ions to move in and out of the neuron.
- To mimic the probabilistic dynamics of the ion channels in the neuron, Hodgkin and Huxley proposed that channels consist of **four components** that can be opened or closed.

- For potassium channels, the four components have identical probability n to be open, resulting in a probability $\mathbb{P}_K = n^4$. Opening and closing of these components depend on the membrane potential V , which determines their transition from open to closed position.

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- Thus, a component passes from the closed (with probability $1 - n$) to the open state (with probability n) following coefficients α_n and β_n , themselves dependent on V ,

$$1 - n \xrightleftharpoons[\beta_n]{\alpha_n} n,$$

obeying the **Boltzmann equations** describing the stochastic behavior of K^+ ions channels and have the general form

$$\alpha_n(V) = \frac{\theta_{n,1}(V - \theta_{n,2})}{\theta_{n,4} + \exp\left(\frac{\theta_{n,2} - V}{\theta_{n,3}}\right)} \quad , \quad \beta_n(V) = \theta_{n,5} \exp\left(-\frac{V}{\theta_{n,6}}\right),$$

where $\theta_{n,i}$, $i = \{1, 2, 3, 4, 5, 6\}$ values are found experimentally (by fine-tuning) to fit the neuron's behavior best.

- If the initial value of the probability n that the K^+ is opened is known, the values of n in time are given by the dynamical system:

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$$n_\infty = \frac{\alpha_n}{\alpha_n + \beta_n} \quad \text{and} \quad \tau_n = \frac{1}{\alpha_n + \beta_n}.$$

n_∞ is the n value of equilibrium and the constant τ_n the approaching time of this equilibrium. As α_n and β_n , these values depend on the membrane potential V . We can then rewrite the equation describing the evolution of n as follows,

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- Let I_K be the number of K^+ ions that flow through the membrane per unit time. In the HH model, I_K is given by,

- where in the last equation of the previous slide, V represents the membrane potential, E_K is the equilibrium potential of the K^+ ions, g_K is conductivity of K^+ ions, and n have been previously defined.

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- Note, however, that, most of the time, some of the K^+ ion channels are blocked, and thus we mostly have g_K and very rarely have g_K^{max} .
- If the membrane potential equals the ion's equilibrium potential ($V = E_K$), there is no circulation, and the current I_K is also zero.
- In the HH model, I_K is given by,

$$I_K = n^4 g_K (V - E_K) \quad (2)$$

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- The probability of a Na^+ channel being open and active is thus $\mathbb{P}_{Na} = m^3 h$, and both m and h depend on membrane potential V .
- Let I_{Na} be the Na^+ flux, .i.e., the amount of Na ions flowing through the membrane. I_{Na} is represented by,

$$I_{Na} = m^3 h g_{Na} (V - E_{Na}), \quad (3)$$

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- As we saw for the evolution of the variable n of the K^+ channels, the transition from the open position (with probability m) or closed (with probability $1 - m$) of each component of the Na^+ channels is given by the V -dependent coefficients α_m and β_m ,

$$1 - m \xrightleftharpoons[\beta_m]{\alpha_m} m,$$

obeying the Boltzmann equations describing the stochastic behavior of the Na^+ channels and have the general form

$$\alpha_m(V) = \frac{\theta_{n,7}(V - \theta_{n,8})}{\theta_{n,10} + \exp\left(\frac{\theta_{n,8} - V}{\theta_{n,9}}\right)} \quad , \quad \beta_m(V) = \theta_{n,11} \exp\left(-\frac{V}{\theta_{n,12}}\right),$$

where $\theta_{m,i}$, $i = \{7, 8, 9, 10, 11, 12\}$ values are found experimentally (by fine-tuning) to fit the neuron's behavior best.

- In a similar fashion, for h , transitions from the active position (with probability h) to the inactive position (with probability $1 - h$) are given by the coefficients α_h and β_h ,

$$1 - h \xrightleftharpoons[\beta_h]{\alpha_h} h.$$

and which obey the **Boltzmann equations** describing the stochastic behavior of the channels and have the general form

$$\alpha_h(V) = \theta_{n,13} \exp\left(-\frac{V}{\theta_{n,14}}\right) \quad , \quad \beta_h(V) = \frac{\theta_{n,15}}{\theta_{n,16} + \exp\left(\frac{\theta_{n,17} - V}{\theta_{n,17}}\right)}$$

where $\theta_{m,i}$, $i = \{13, 14, 15, 16, 17\}$ values are found experimentally (by fine-tuning) to fit the neuron's behavior best.

- In the same way as for n , the evolutions of m and h are given by the differential equations,

$$\begin{aligned}\frac{dm}{dt} &= \alpha_m(1 - m) - \beta_m m = \frac{m_\infty - m}{\tau_m}, \\ \frac{dh}{dt} &= \alpha_h(1 - h) - \beta_h h = \frac{h_\infty - h}{\tau_h}.\end{aligned}$$

- From where we extract the following notations:

$$\begin{aligned}m_\infty &= \frac{1}{\alpha_m + \beta_m}, & \tau_m &= \frac{1}{\alpha_m + \beta_m}, \\ h_\infty &= \frac{1}{\alpha_h + \beta_h}, & \tau_h &= \frac{1}{\alpha_h + \beta_h}.\end{aligned}$$

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- Let I_L be the Cl^- flux, .i.e., the amount of Cl^- ions flowing through the membrane. I_L is represented by,

$$I_L = g_L(V - E_L), \quad (4)$$

where V is the membrane potential and E_L is the equilibrium potential of the leakage ions (mostly Cl^-).

- From the conservation of electric charges, **Kirchhoff's current law** applied to the circuit in the Figure 3 given slide 4 above

$$I = I_0 = C \frac{dV}{dt} + I_K + I_{Na} + I_L,$$

- Where I_0 is the input current, I_K is the potassium current, I_{Na} is the sodium current, I_L is the leak current, which models all ions that flow through the ion channels, which are always open, the capacitance of the membrane is represented by C_m , and V represents the voltage of the membrane potential. We therefore have

$$-C_m \frac{dV}{dt} = I_K + I_{Na} + I_L - I_0,$$

which, after dropping the subscript m in C_m , is given more explicitly by

$$-C \frac{dV}{dt} = n^4 \bar{g}_K (V - E_K) + m^3 h g_{Na} (V - E_{Na}) + \bar{g}_L (V - E_L) - I_0.$$

- Thus, the full model of the nerve impulse proposed by Hodgkin and Huxley is

$$\left\{ \begin{array}{l} C \frac{dV}{dt} = n^4 g_K (E_K - V) + m^3 h g_{Na} (E_{Na} - V) + g_L (E_L - V) + I_0, \\ \frac{dn}{dt} = \alpha_n (1 - n) - \beta_n n = \frac{n_\infty - n}{\tau_n}, \\ \frac{dm}{dt} = \alpha_m (1 - m) - \beta_m m = \frac{m_\infty - m}{\tau_m}, \\ \frac{dh}{dt} = \alpha_h (1 - h) - \beta_h h = \frac{h_\infty - h}{\tau_h}. \end{array} \right.$$

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- Where t is the time in ms. Note that conductances g_K and g_{Na} do not remain constant: they depend on V (in a very complicated way). They are both only constant when the corresponding ion channels are all opened, in which case g_K and g_{Na} becomes g_K^{max} and g_{Na}^{max} , respectively. g_L is a constant. (Why is it always a constant?)

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- The so-called gating variables m , n , h are all bounded in the unit interval $[0, 1]$. (Why do you think it is so?)

- The Boltzmann equations give the expressions of the **so-called rate constants** of the HH neuron model, $\alpha_j(V)$ and $\beta_j(V)$, where $j = \{n, m, h\}$, together with the values of the other parameters $\theta_{j,i}$, are determined experimentally by using the Voltage Clamp experiments.

$$\alpha_n(V) = \frac{0.01(10 - V)}{\exp\left(\frac{10 - V}{10}\right) - 1}, \quad \alpha_m(V) = \frac{0.1(25 - V)}{\exp\left(\frac{25 - V}{10}\right) - 1}$$

$$\alpha_h(V) = 0.07 \exp\left(\frac{-V}{20}\right), \quad \beta_n(V) = 0.125 \exp\left(\frac{-V}{80}\right),$$

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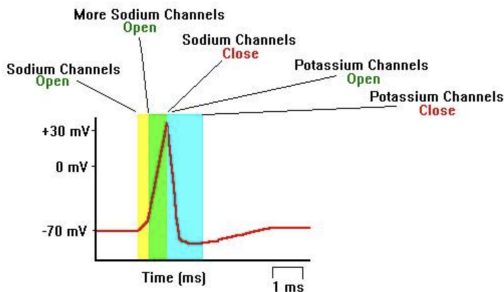
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$$C = 1.0 \mu F/cm^2, \quad E_{Na} = 115.0 mV, \quad E_K = -12.0 mV,$$

$$E_L = 10.6 mV, \quad g_{Na} = g_{Na}^{max} = 120.0 mS/cm^2,$$

$$g_K = g_K^{max} = 36.0 mS/cm^2, \quad g_L = g_L^{max} = 0.3 mS/cm^2$$

- In the absence of external current I_0 : equilibrium.
- After a weak impulse from I_0 : rapid relaxation to equilibrium.
- After a sufficiently strong (short) from impulse I_0 : Fires for the corresponding short duration.
- With constant, sufficiently strong I_0 : The pulses occur periodically (**limit cycle!**)
- Immediately after repolarization, the HH neuron cannot fire again immediately (**refractory period**).



Profile of a spike of the HH neuron

$$\left\{ \begin{array}{l} C \frac{dV}{dt} = n^4 g_K (E_K - V) + m^3 h g_{Na} (E_{Na} - V) + g_L (E_L - V) + I_0, \\ \frac{dn}{dt} = \alpha_n (1 - n) - \beta_n n = \frac{n_\infty - n}{\tau_n}, \\ \frac{dm}{dt} = \alpha_m (1 - m) - \beta_m m = \frac{m_\infty - m}{\tau_m}, \\ \frac{dh}{dt} = \alpha_h (1 - h) - \beta_h h = \frac{h_\infty - h}{\tau_h}. \end{array} \right.$$

- Dynamics of the HH equations are relatively simple. However, their explicit analysis (fixed points (equilibria), stability, bifurcations, etc) is extremely difficult because of the “unwieldy” right-hand sides, (i.e., strong nonlinearity, high dimensionality, & large parameter space.)

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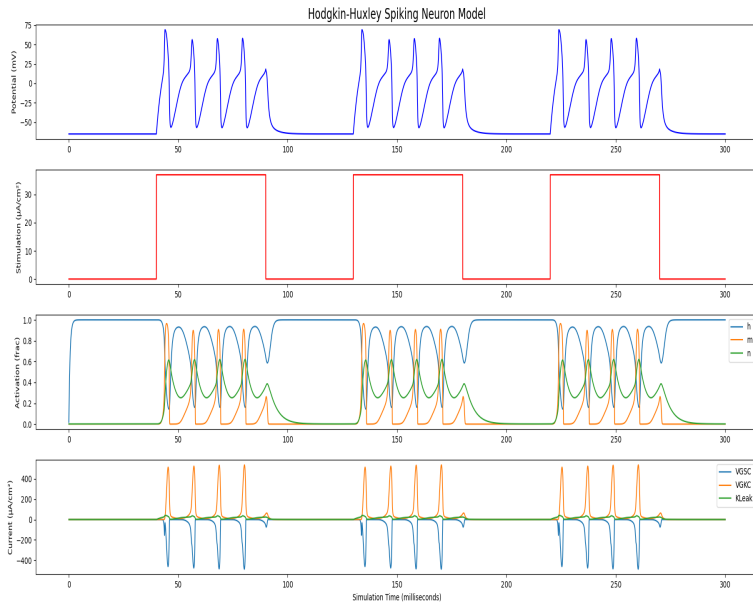
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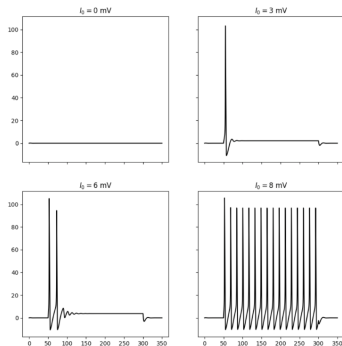
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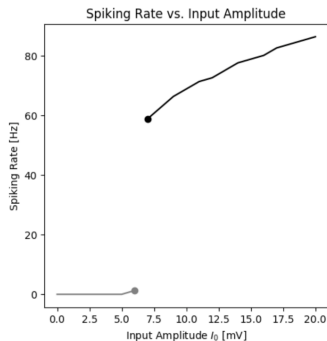
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- Thus, one would like to have the system of equations of V made up of simpler equations but with similar properties to the HH model.
- This can be via timescale separation analysis and exploiting similarities between the HH variables.



- By simulating the neuron, we can observe that the membrane potential begins to generate action potentials (**spikes**) once the input current increases above a certain threshold.
- As shown below, we can visualize the membrane potential V at different input currents. Additionally, we can look at the steady-state spiking rate (in Hz) of the HH neuron.



Responses of the simulated neuron at different input currents.

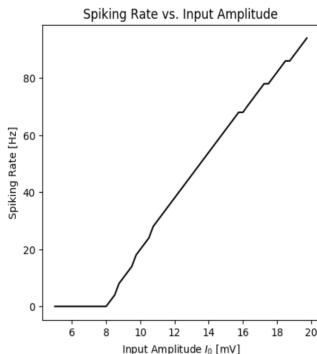
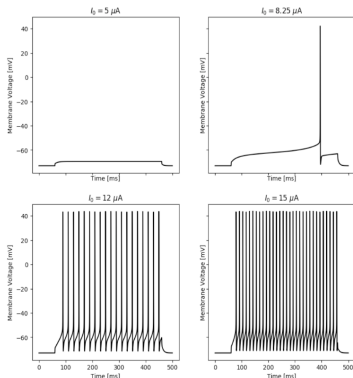


Spiking rate of a neuron as a function of the input current.

Firing rate (spiking rate) as a function of the input current I_0 in Hodgkin-Huxley (HH) neuron model and ReLU-type behavior.

[M. Yamakou]

- By simulating the neuron, we can observe that the neuron's firing rate is a function of the input current. In this standard model, the firing rate $r(I_0)$ could be approximated by a **Heaviside function**. However, different types of functions can be obtained by different neuron types.



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- This abstraction is used to move from the complex and computationally expensive model to a simpler and cheaper **rate-based models**. As a result, it is easier to model networks of neurons. This transition has formed the basis for today's popular **artificial neural networks (ANN)**.