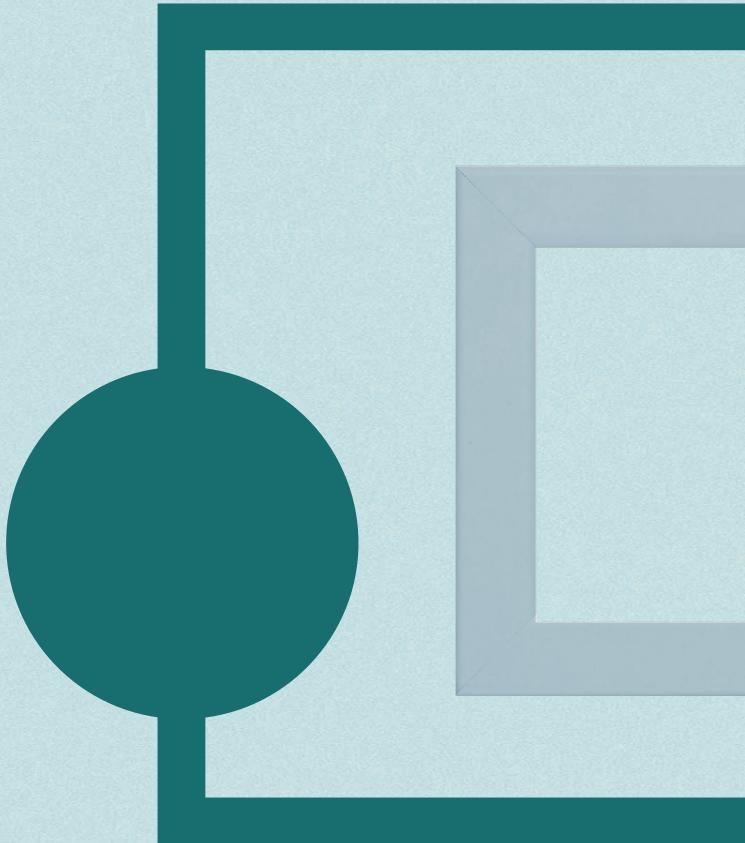
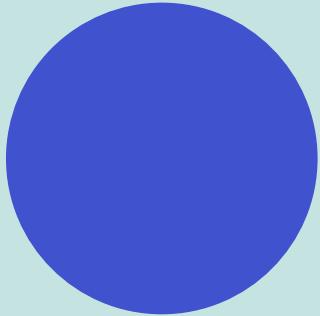


Neuro B2: Excitatory- Inhibitory Pair

By Shaivi Nandi, Ruthvik CSS,
Utkarsh Goyal, Navya Balaji





01

A Biological Background

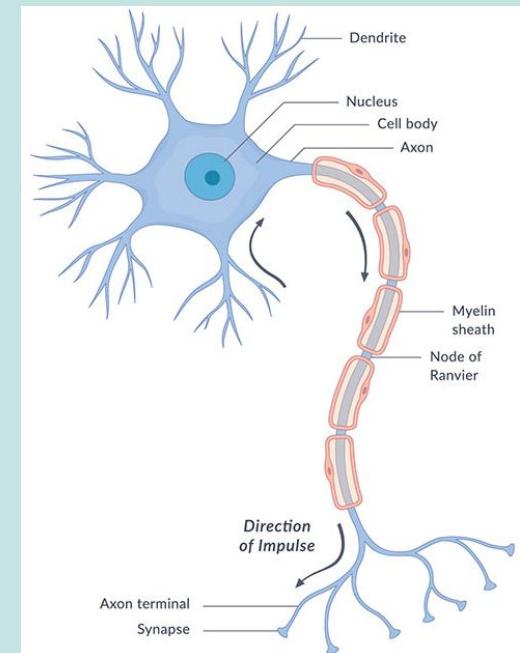
Generation and Transmission of
Nerve Signals



Nerve Structure

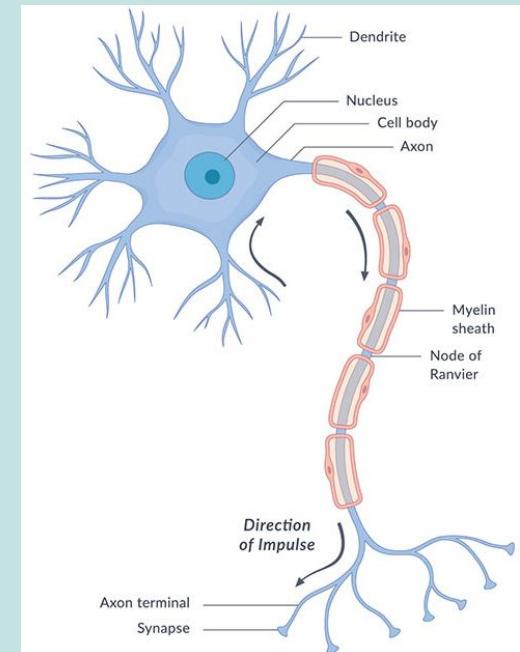
1. **Dendrite** - Receives and integrates multiple incoming signals from neurons through receptor on its ends.
2. **Axon** - The long part of the neuron through which the signal propagates quickly and covers a long distance.
3. **Synapse** - The part where ions / molecules that act as neurotransmitters are released acting as messengers for the next neuron.

Dendrite -> Axon -> Synapse



Nerve Structure

4. **Myelin Sheath** - Fatty insulating layer wraps around the axon, preventing ion leakage and allowing the signal to travel faster, just like insulation on a wire.
5. **Axon Hillock** - The decision center of the neuron where all incoming excitatory and inhibitory signals are combined; if they add up to exceed the threshold, an action potential is triggered.

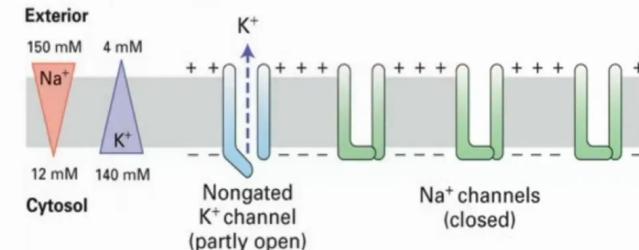


What happens inside a neuron?

1. Inside the cell
 - a. Conc. of K+ 
 - b. Conc. of Na+ 
2. Outside the cell
 - a. Conc. of K+ 
 - b. Conc. of Na+ 
3. The passage of K+ is kept slightly open causing imbalance of charges.
4. What happens if we open Na+ gates? How do we “repolarize”?

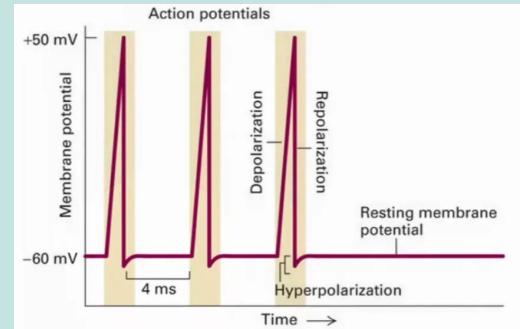
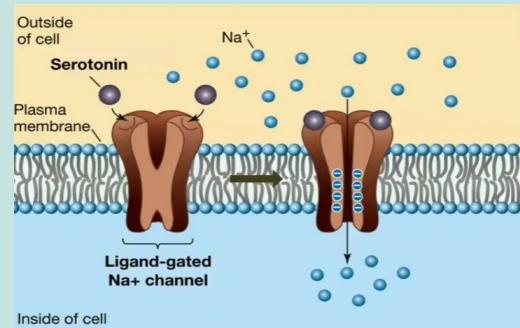
Negative membrane potential results from:
1) leaky K+ channels
2) K+ moving down its concentration gradient

(a) Resting state (cytosolic face negative)



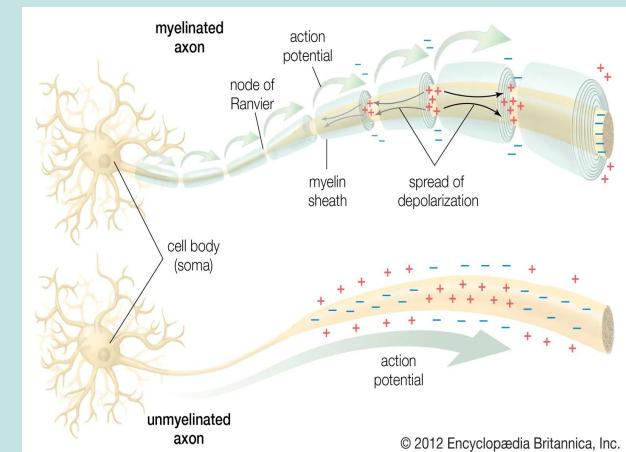
Generation of Nerve Impulses

1. Neurotransmitters bind to their respective receptors signalling the corresponding channels to open.
2. This opening of channel causes “depolarization” or “repolarization” of the membrane.
3. How is the “strength” of the signal decided? How is it controlled?



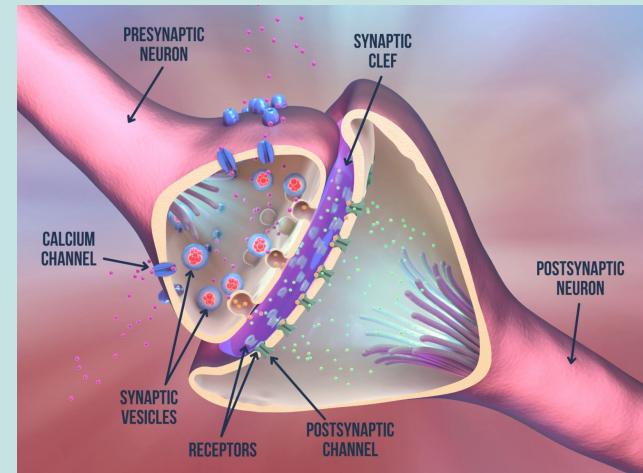
Propagation of action potential

1. The axon is insulated by “myelin” sheaths. That means there are no Na^+ or K^+ channels in that region.
2. There are gaps in the sheath known as “Nodes of Ranvier”.
3. The voltage is regenerated at each of these nodes exponentially increasing the speed of transmission.
4. But how and why?



Communication Between Neurons

1. Action potential arrives → triggers calcium ions to enter presynaptic neuron
2. Calcium causes vesicles to release neurotransmitters into synaptic cleft
3. Neurotransmitters bind to receptors on postsynaptic neuron
4. Receptor type determines: Excitatory or Inhibitory signal

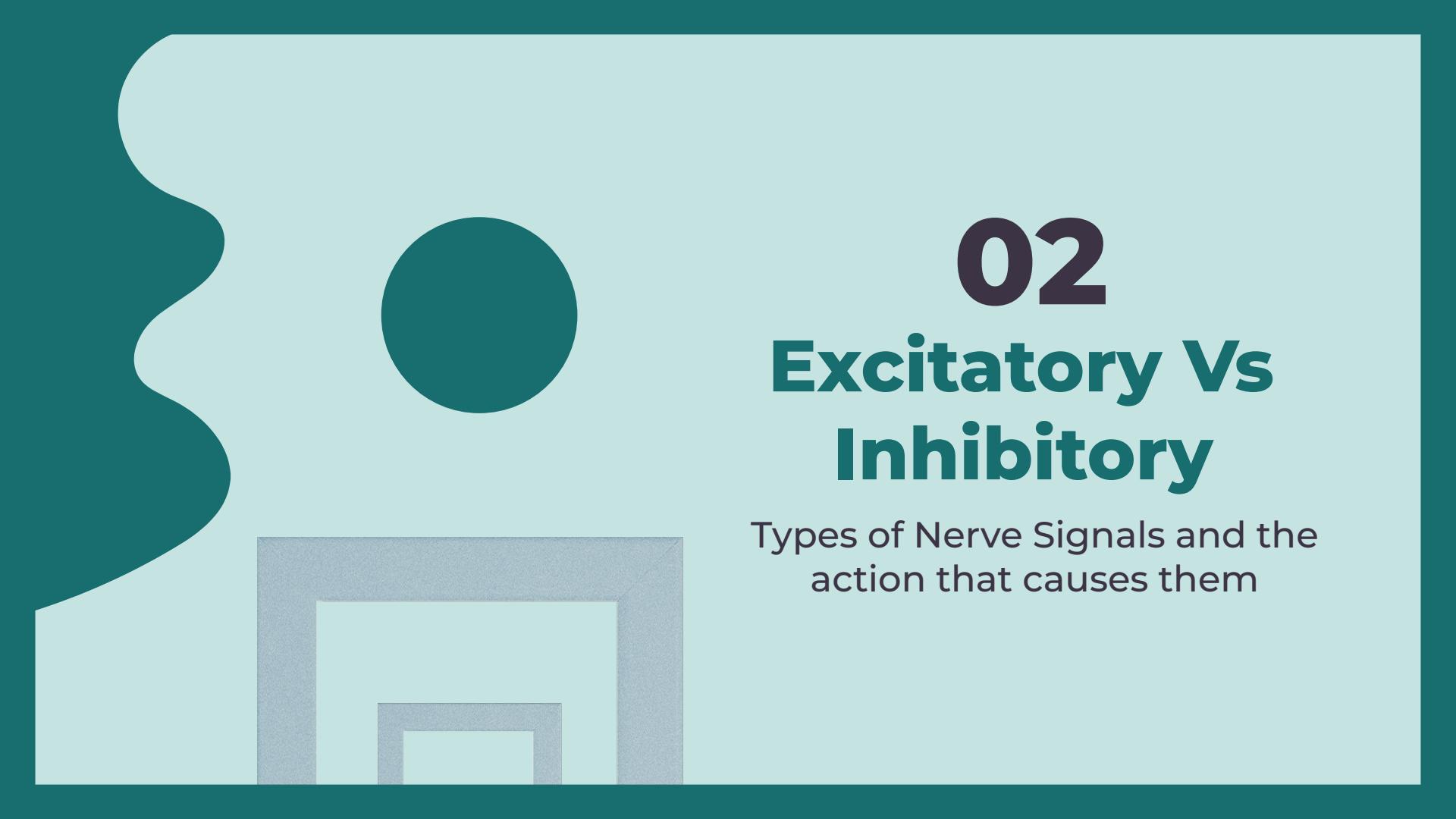


Excitatory Synapse

1. Excitatory neurotransmitters (glutamate) released
2. Positive ions (Na^+) enter postsynaptic neuron
3. Membrane potential becomes more positive
4. Postsynaptic neuron fires action potential → signal passes forward ✓

Inhibitory Synapse

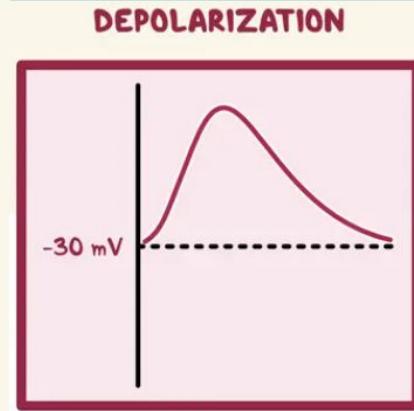
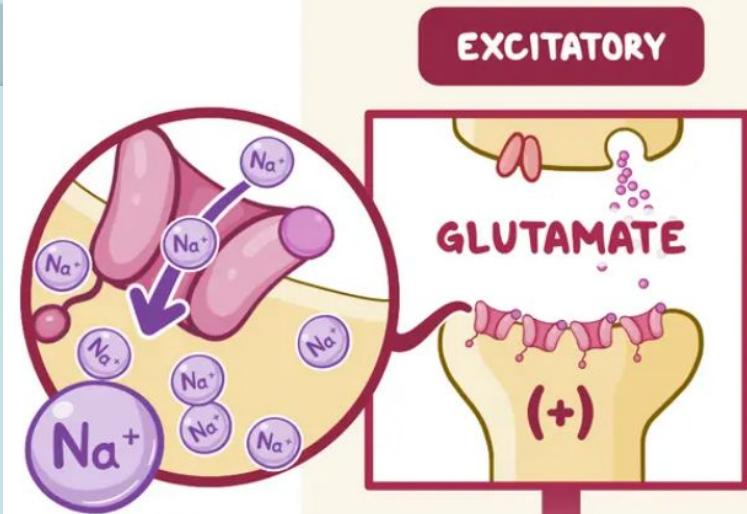
1. Inhibitory neurotransmitters (GABA, glycine) released
2. Negative ions (Cl^-) enter OR positive ions (K^+) exit postsynaptic neuron
3. Membrane potential becomes more negative
4. Postsynaptic neuron less likely to fire → signal is blocked ✗



02

Excitatory Vs Inhibitory

Types of Nerve Signals and the
action that causes them

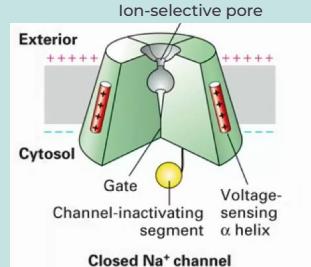


Propagation of Excitatory signal

Excitatory Neurotransmitter Release

01

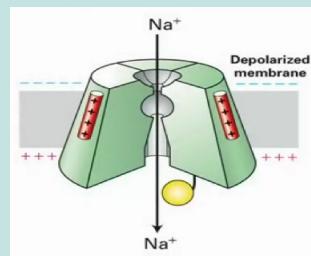
Excitatory neurotransmitters (Ex. Serotonin) bind to ligand-gated Na^+ channels on the postsynaptic membrane.



02

Opening of ion channel

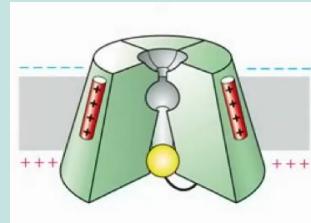
Na^+ channels open, sodium rushes in, causing depolarization.



03

Action potential threshold

Membrane crosses threshold; voltage-gated Na^+ channels open and create the action potential.



Propagation of Excitatory signal

0
4

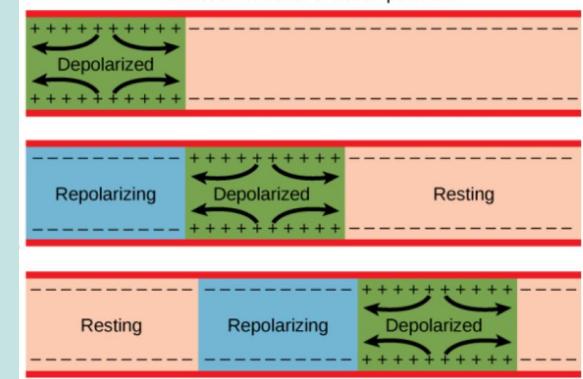
Axon propagation

The Action Potential travels with great speed along the axon. Unidirectionality is maintained by ion channel inactivation.

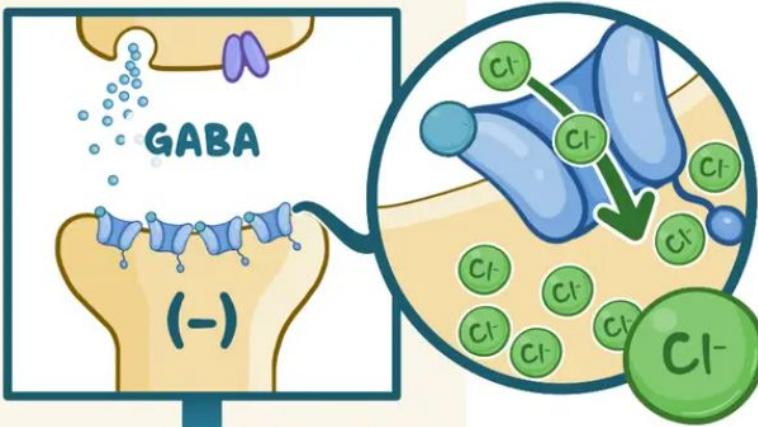
05

Repolarization

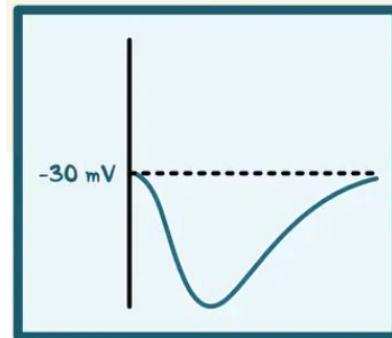
K^+ leaves the cell down its gradient, and its channel opens later than Na^+ due to a naturally slower protein structure.



INHIBITORY



HYPERPOLARIZATION



Propagation of Inhibitory signal

01

Inhibitory Neurotransmitter

Release

An inhibitory neurotransmitter(Ex. glycine) is released that activate Cl^- or K^+ channels on the postsynaptic membrane.

02

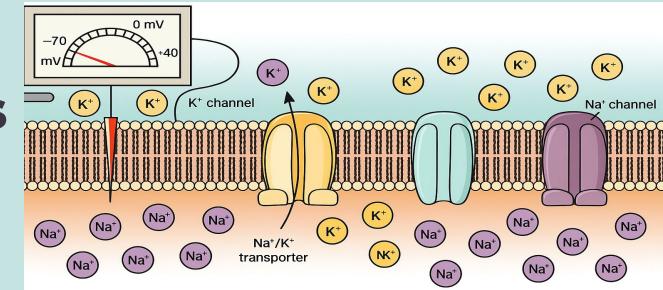
Opening of Cl^- / K^+ Channels

K^+ ions are let out of the cytoplasm. Also implying the same as letting Cl^- ions inside.

03

Hyperpolarization of the Membrane

Making the inside of the membrane more negative causes hyperpolarization.



Propagation of Inhibitory signal

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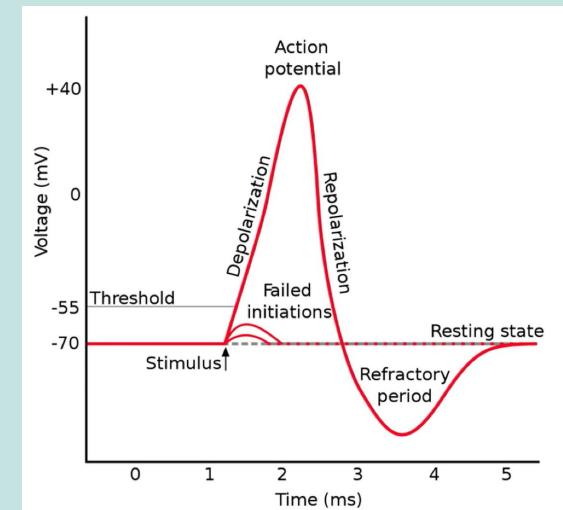
Shunting of Excitatory Inputs

Excitatory signals are counteracted upon thus reduced the chances of reaching the axon hillock and triggering action potential.

05

Refractory Recovery Phase

Restoration of resting potential; K⁺ and Cl⁻ ion channels close, resting potential is re-established. Sodium-Potassium conc are reset.



Excitatory and Inhibitory signal at axon hillock

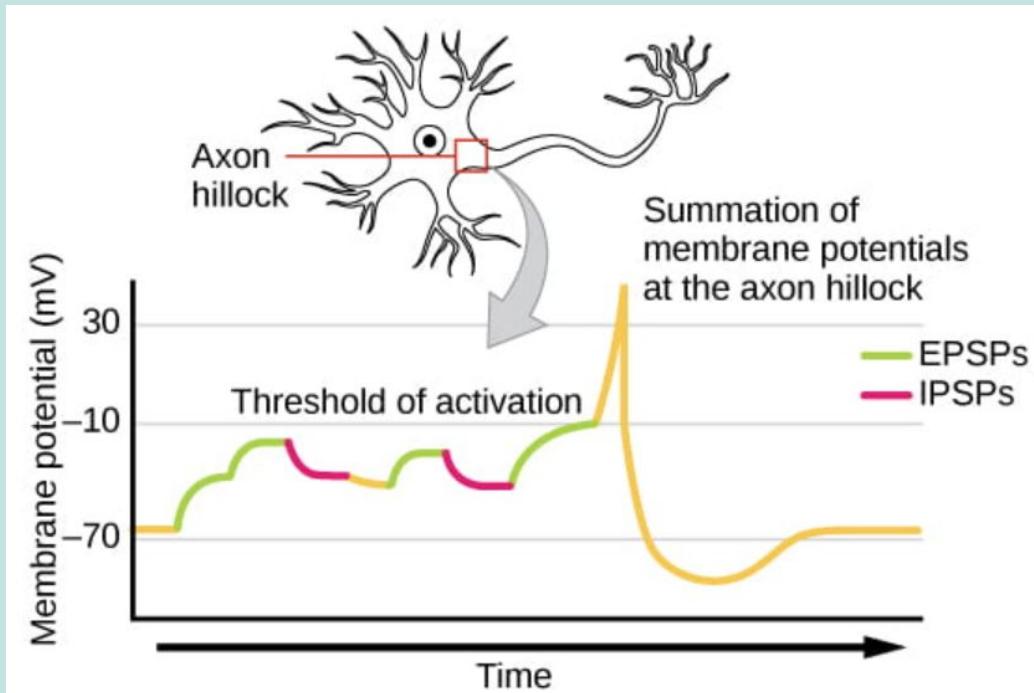
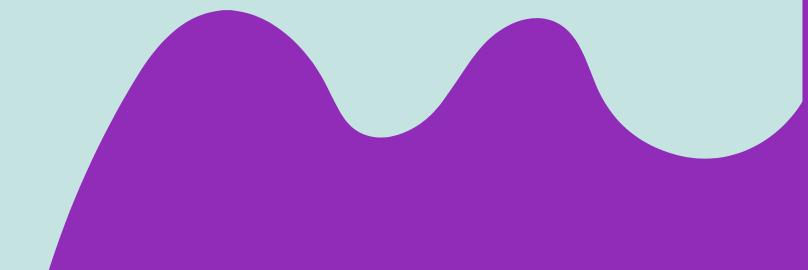
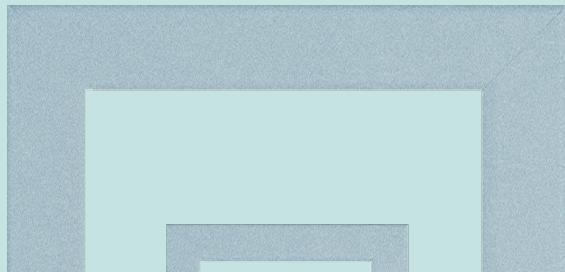
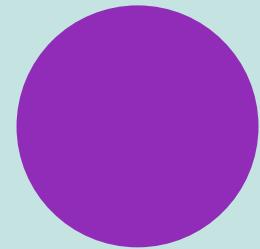


Fig. Summation of EPSPs and IPSPs at the Axon Hillock

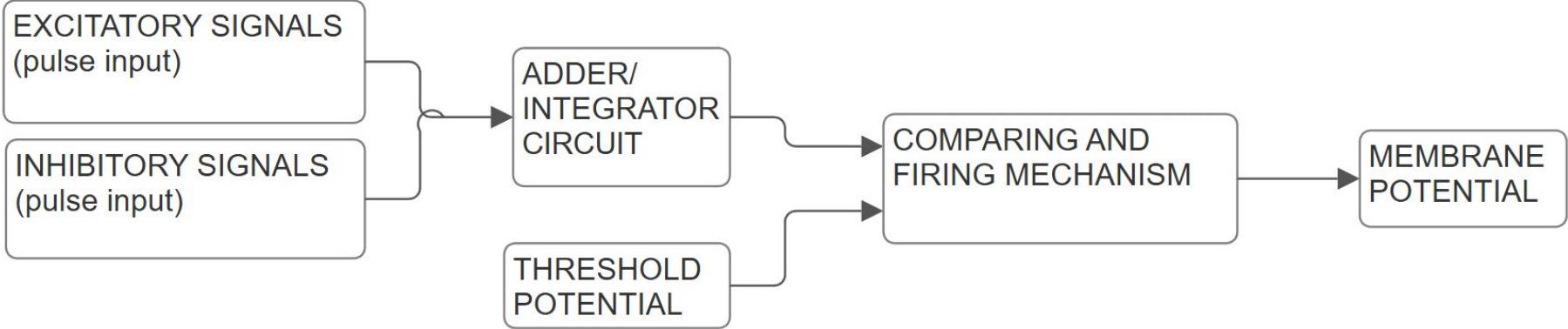
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Electronic Equivalents

Modelling the nerve using basic electronic components.



BLOCK DIAGRAM



PASSIVE ELEMENTS THAT CAN BE USED

Capacitor:

- Models the cell membrane and stores charge
- Integrates excitatory and inhibitory inputs over time



Resistors:

- Control current flow and synaptic resistance
- Set the integration time constant



Switches:

- Model synaptic ion channels opening/closing
- Enable/disable excitatory and inhibitory pathways

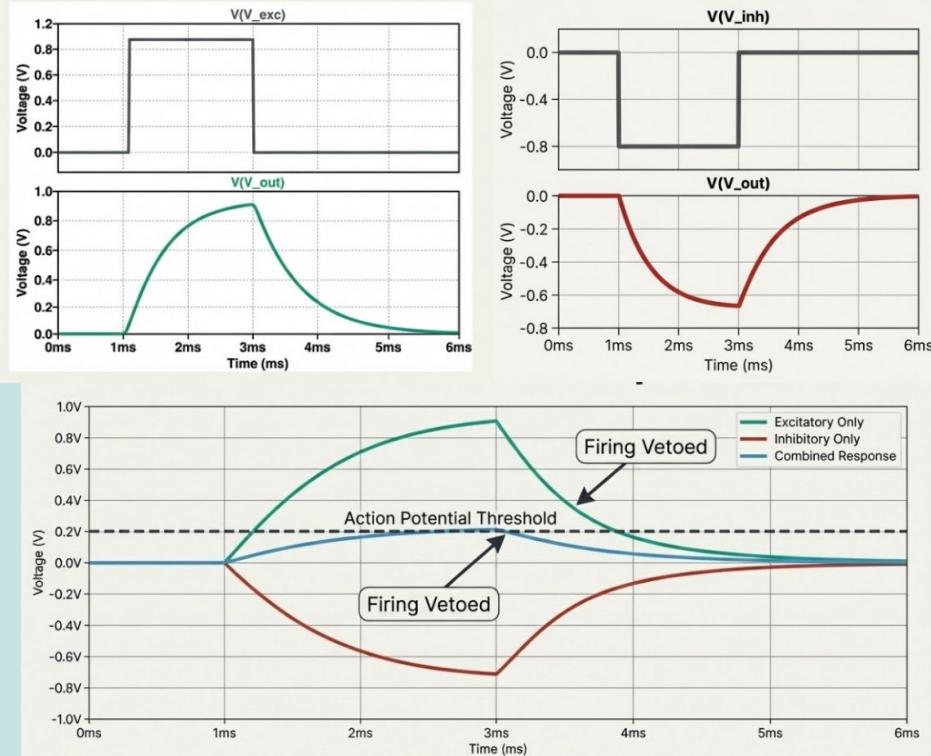


Voltage Sources:

- Represent presynaptic electrochemical driving forces
- Create differential excitation/inhibition on the membrane



PASSIVE ELEMENTS THAT CAN BE



Why Passive Elements Alone Cannot Generate Action Potentials

No Threshold Detection:

- Passive circuits respond proportionally to inputs; they cannot detect threshold

No Regenerative Feedback:

- Action potentials need amplification, but passive components only store/dissipate energy passively

Cannot Reset Automatically:

- Passive R-C circuits decay exponentially; they cannot actively repolarize the membrane as real neurons do

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