NEUROSYNC-

Neuro-B5: Adaptive Neuron (Self-Inhibiting RC)

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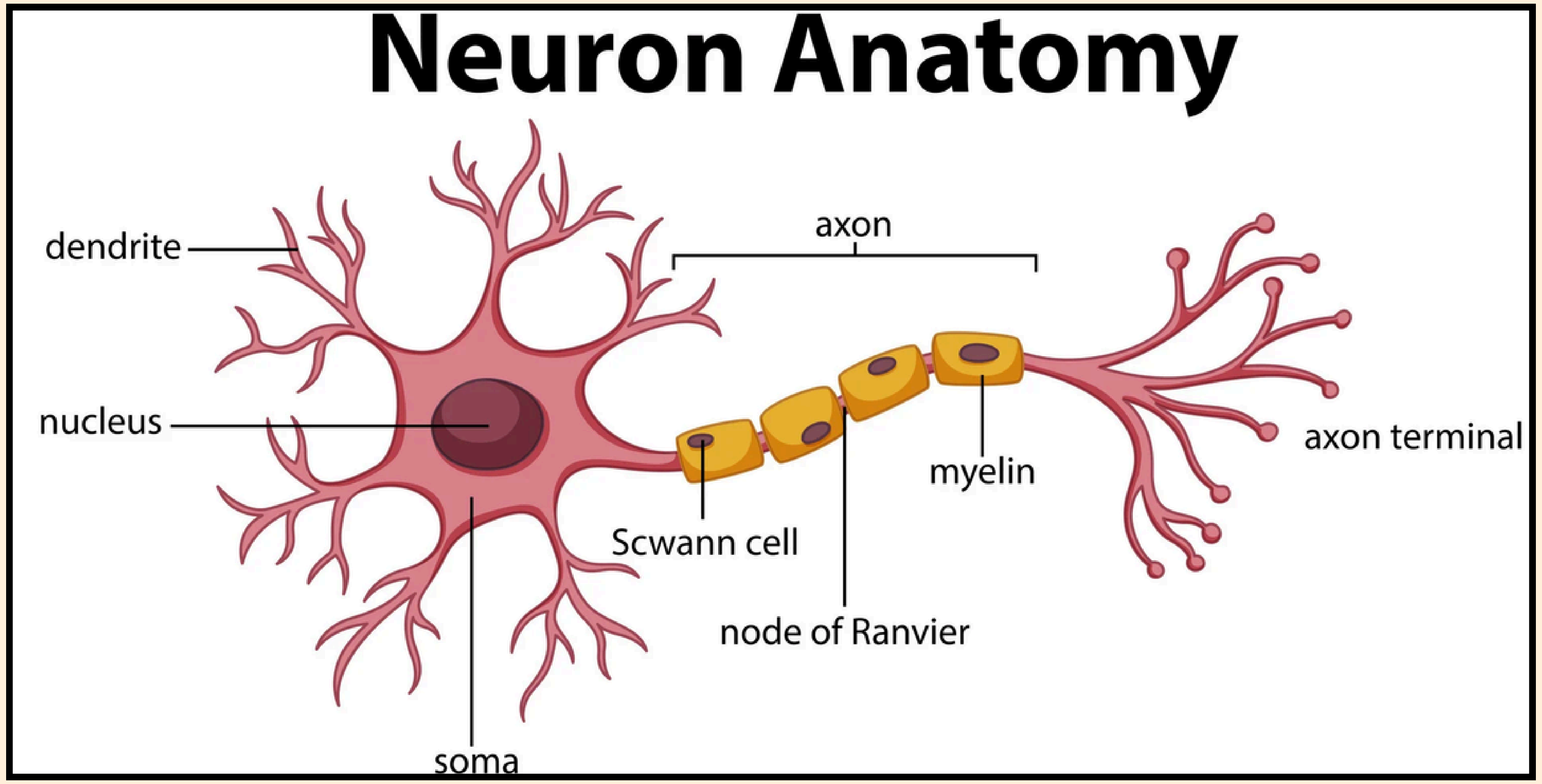
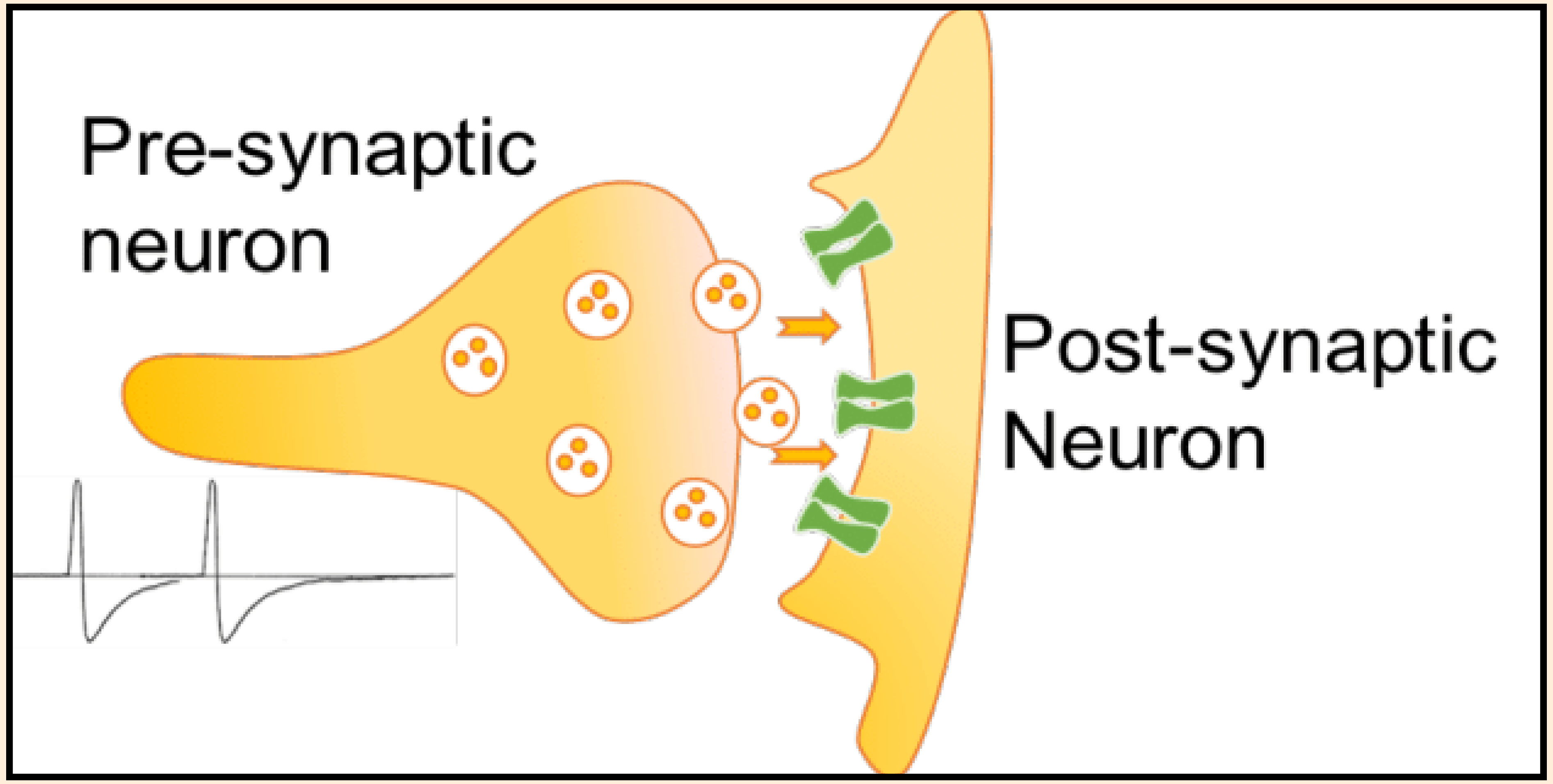
Neuron and its Terminologies-

1. Dendrites : Branch-like structures that receive signals from other neurons. Bring electrical/chemical information toward the cell body.
2. Cell Body (Soma) : Contains the nucleus and basic cell machinery. Integrates incoming signals and decides whether to generate a spike.
3. Axon : a long cable-like extension that carries electrical impulses (action potentials) away from the neuron to other cells.



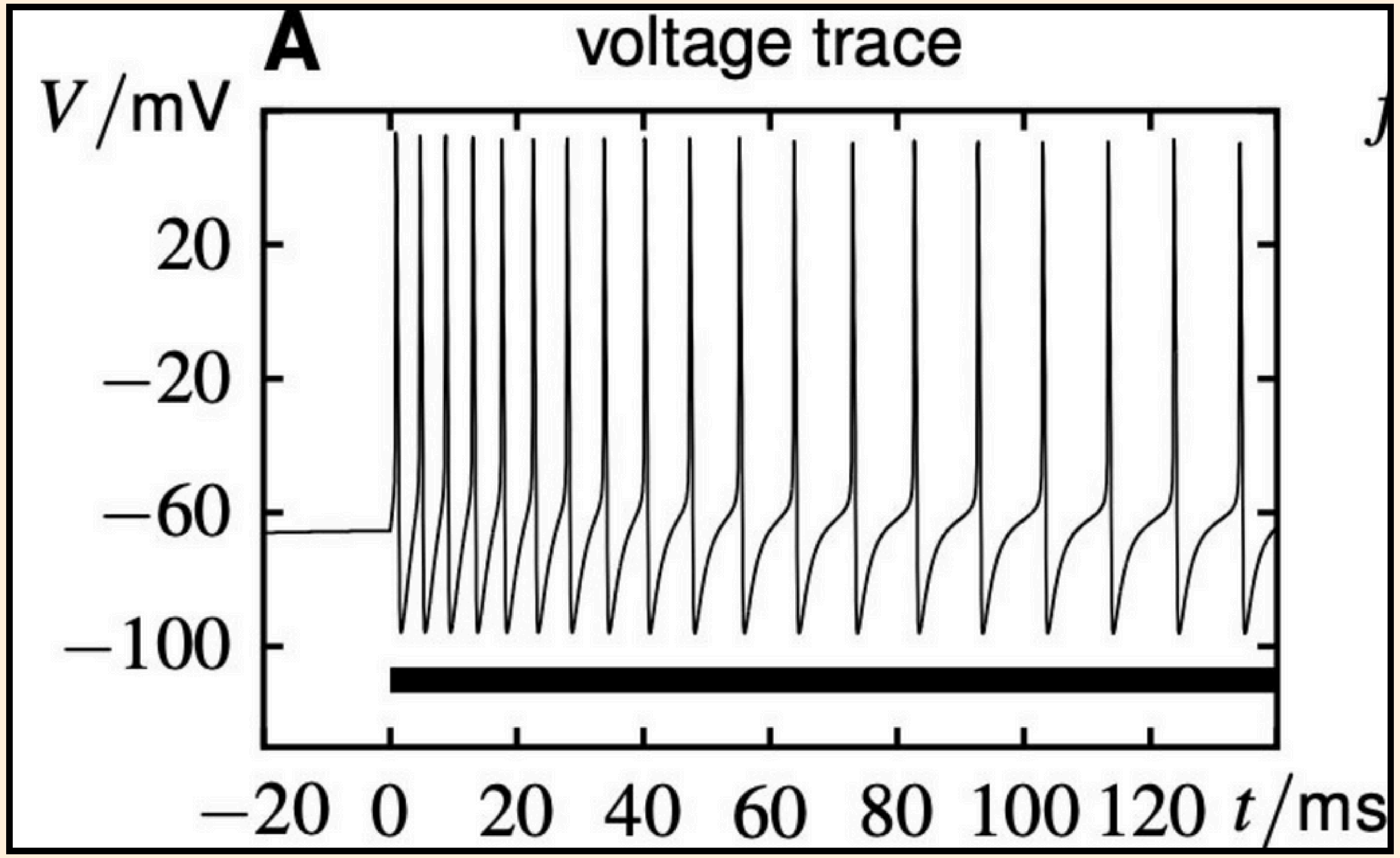
SYNAPSES AND ACTION POTENTIAL

- A synapse is the tiny junction where one neuron sends chemical signals (neurotransmitters) to another, influencing whether it will fire.
- An action potential is a rapid, all-or-nothing electrical spike produced when the neuron's membrane voltage crosses a threshold.
- The action potential travels down the axon by triggering nearby ion channels in sequence, creating a wave of depolarization that moves like falling dominoes along the axon.



NEURAL ADAPTATION / SELF INHIBITION

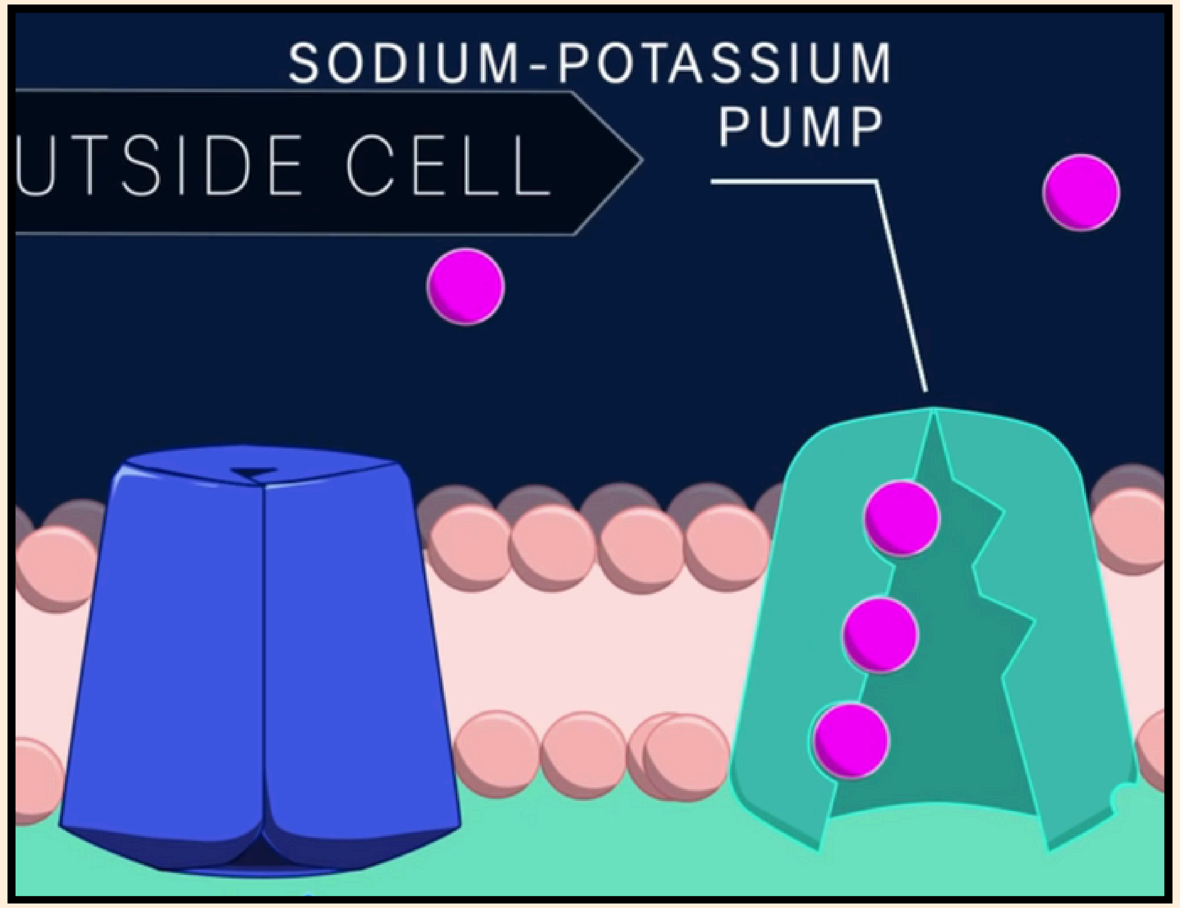
- Neural adaptation is when a neuron reduces its firing rate over time even though the input stimulus stays constant.
- This happens because slow ion channels (often potassium or calcium-dependent) activate during repeated firing and make the neuron less excitable.
- As a result, each spike increases a brief self-inhibition, causing the neuron to fire rapidly at first and then gradually slow down.

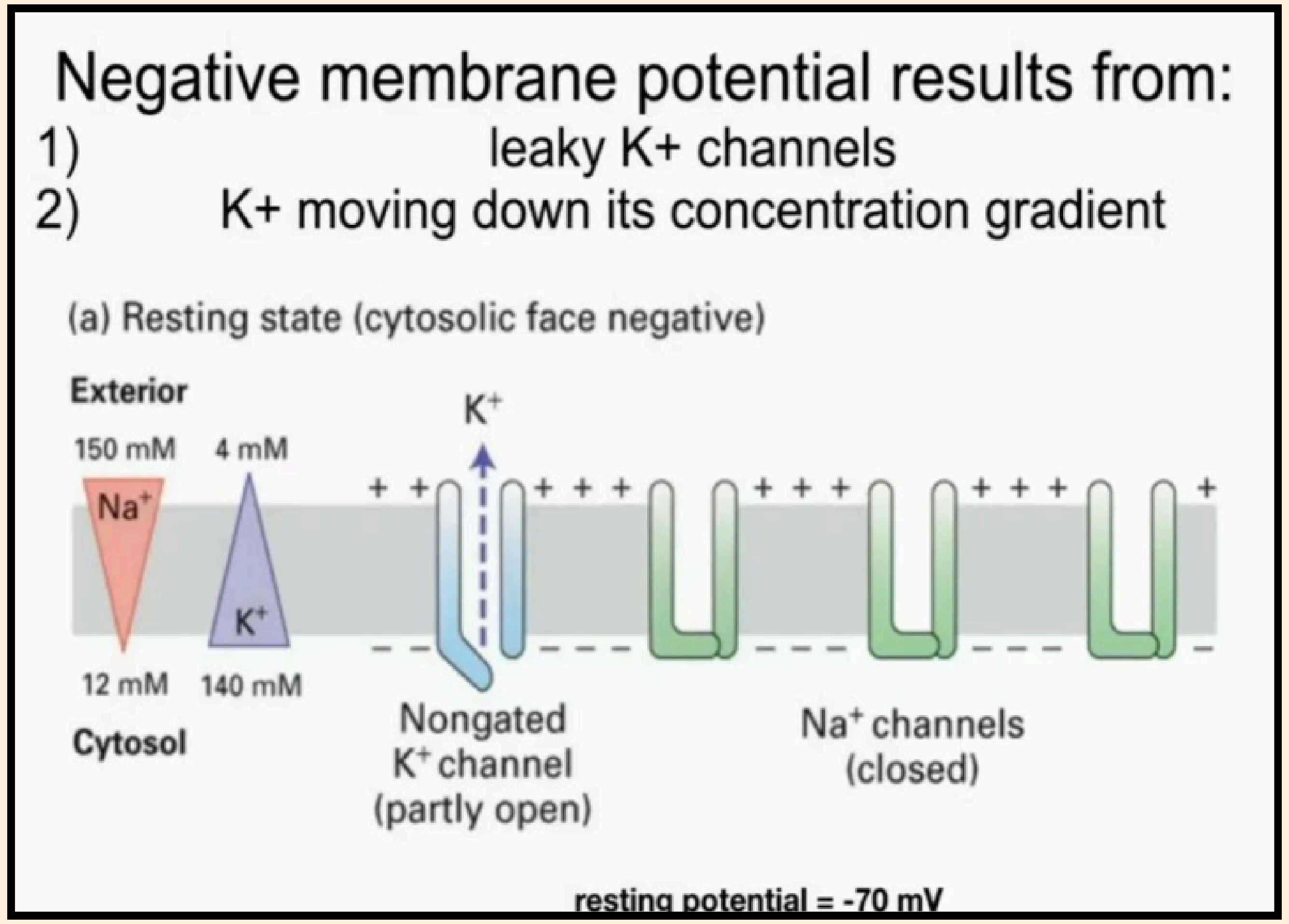


THE LEAKY INTEGRATE-AND-FIRE NEURON MODEL

SODIUM-POTASSIUM PUMP

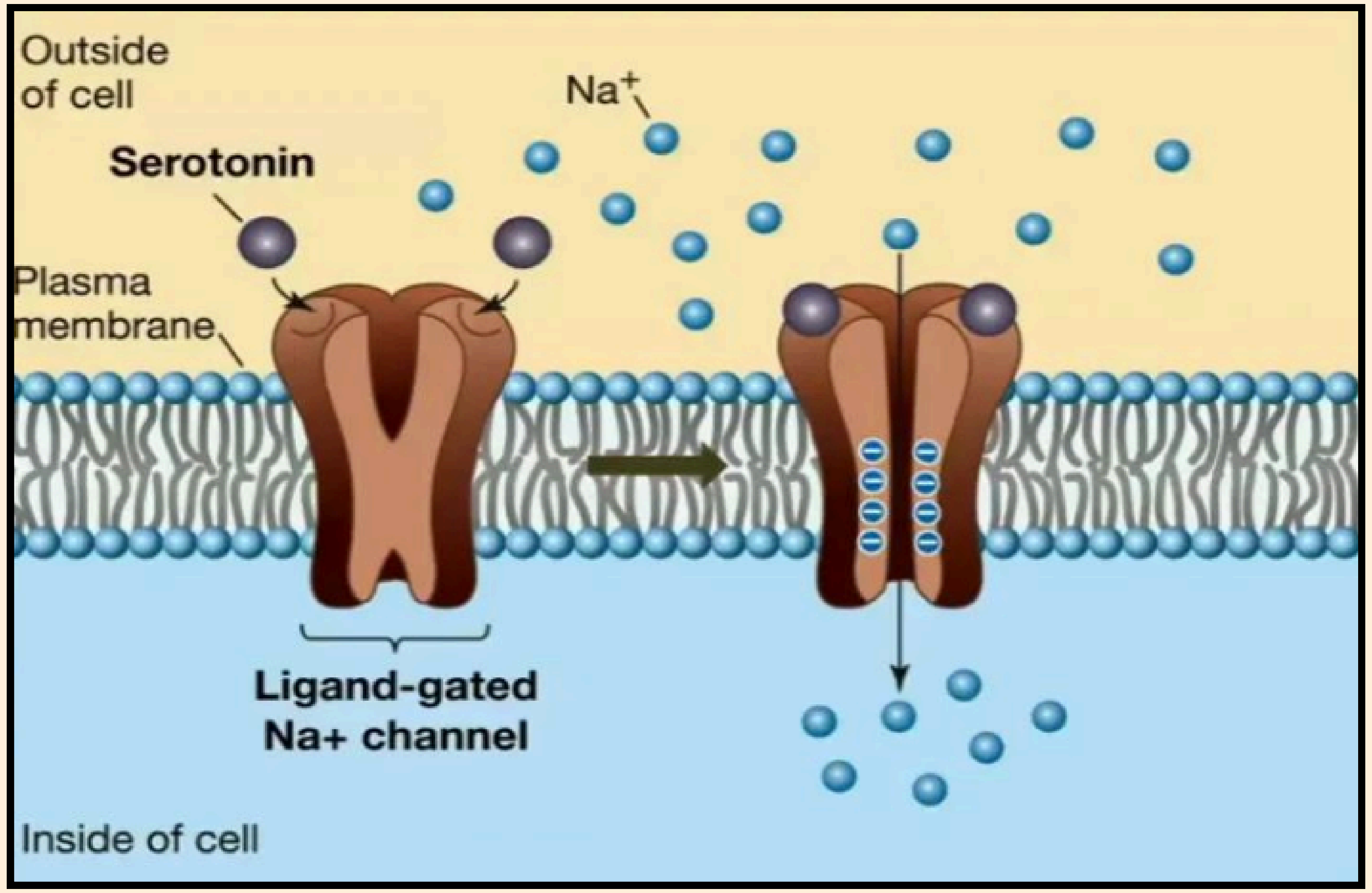
- Initially, there are equal concentrations of Na+ and K+ ions on both sides of the membrane. Still, the sodium-potassium ATPase hydrolyzes ATP and uses its energy to transfer 3 Na+ ions outside the membrane in return for 2 K+ ions inside the membrane.
- This, along with a Leaky Nongated K+ channel, leads to a higher concentration of K+ ions inside and Na+ ions outside, leading to a net negative potential across the membrane, also called the membrane potential (70 mV)





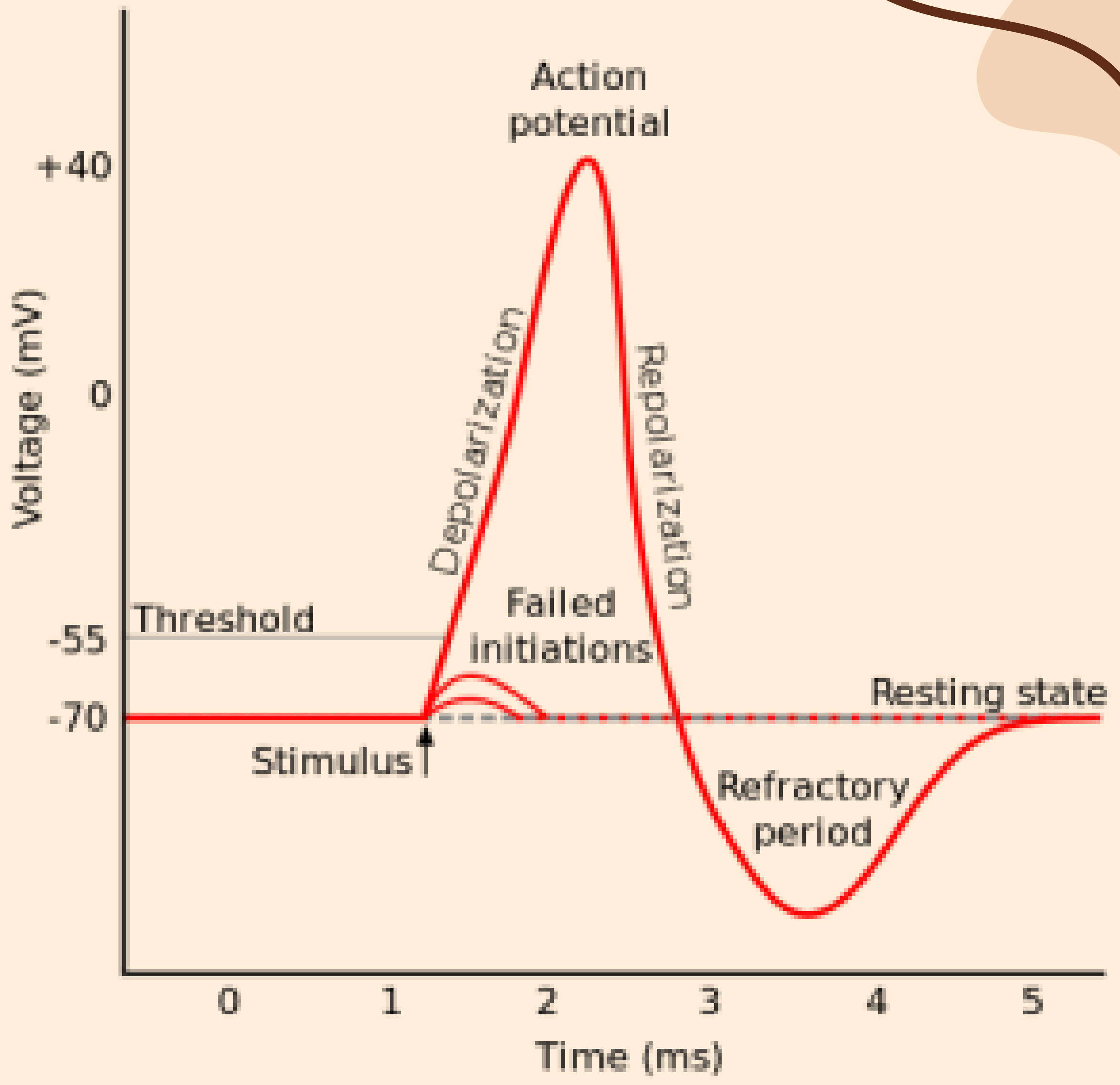
LIGAND AND VOLTAGE CHANNELS-

- There are essentially two types of Na+ channels, the ligand-dependent channel will open or close depending upon the neurotransmitter that is attached to it. If the transmitter is excitatory, the channel will open and increase potential or else it will close and decrease potential.
- The voltage-dependent channel will only open when the membrane potential has reached a certain threshold potential (-50 to -55 mV)



SPIKING AND POLARIZATION

- Depolarization: when Na+ ions enter the membrane, thus increasing the potential.
- Polarization: after some time, when the Na+ channels close, the K+ channels open, leading to the outflow of K+ ions, thus decreasing the potential.
- Hyperpolarization: the K+ channels take a lot of time to close thus leading to excess K+ outflow and hence a potential even below threshold potential (- 70mV).



ADAPTATION IN LIF NEURON MODEL

ADAPTATION

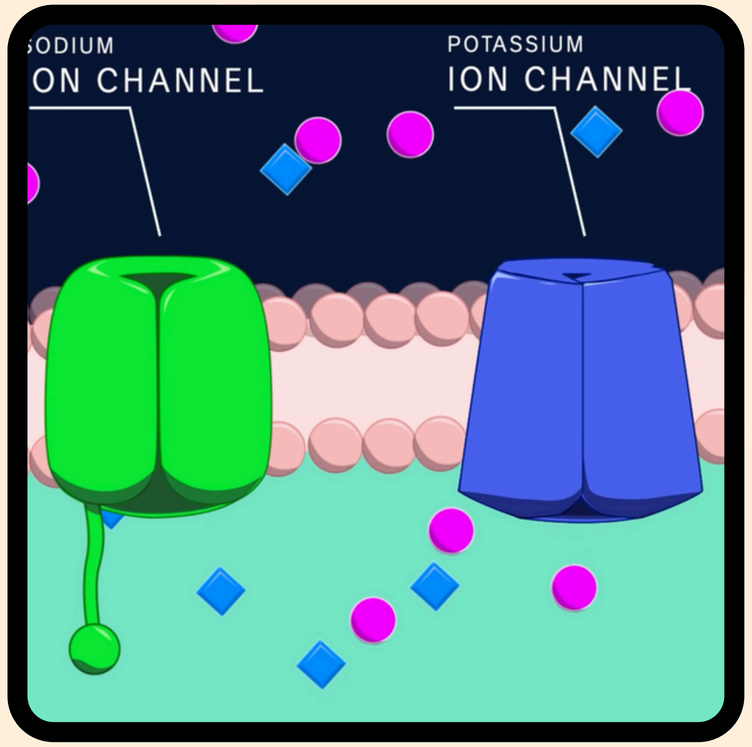
High-frequency firing upon stimulus onset due to minimal adaptation current.

Inter-spike intervals lengthen as adaptive processes accumulate.

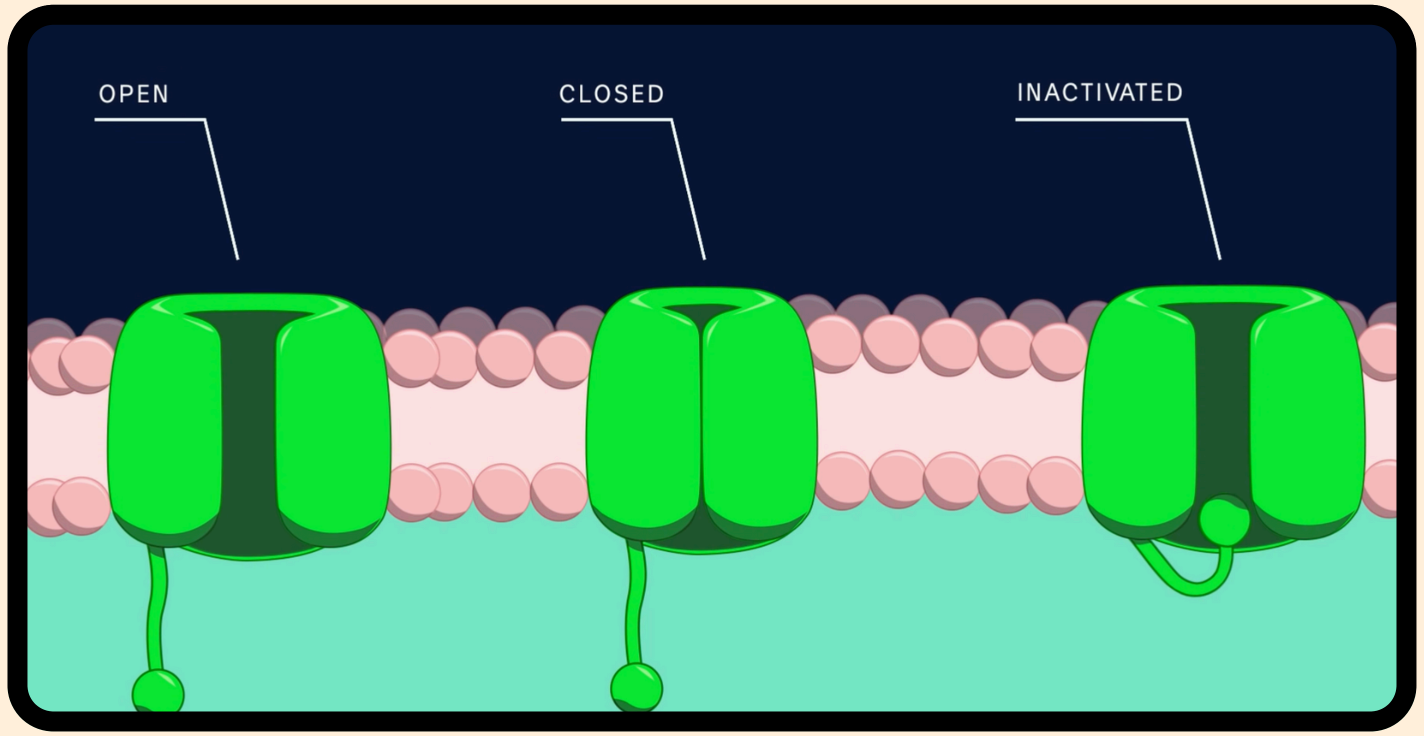
The neuron becomes progressively harder to excite for subsequent spikes.

ADAPTATION METHODS

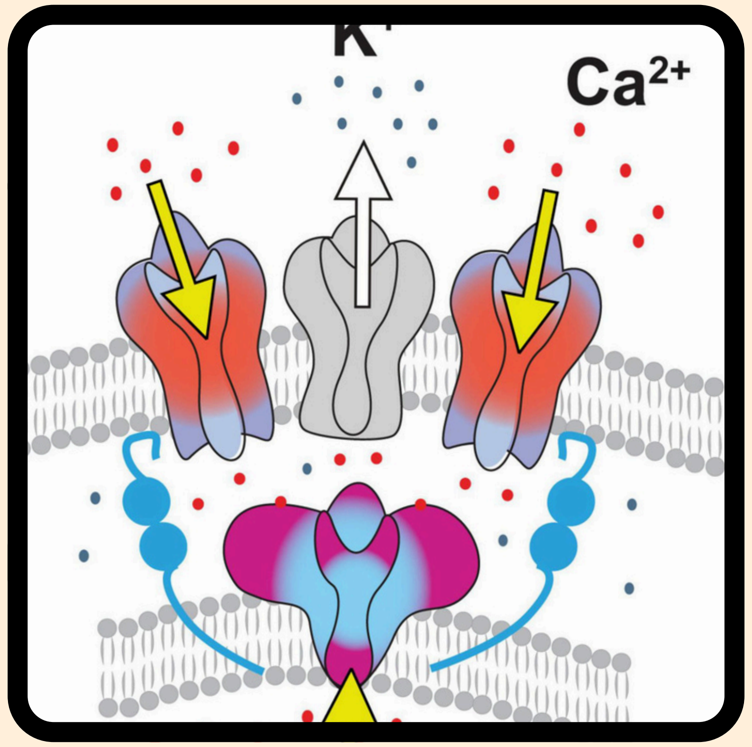
IM Current



Slow Recovery of Na+ ion Channel

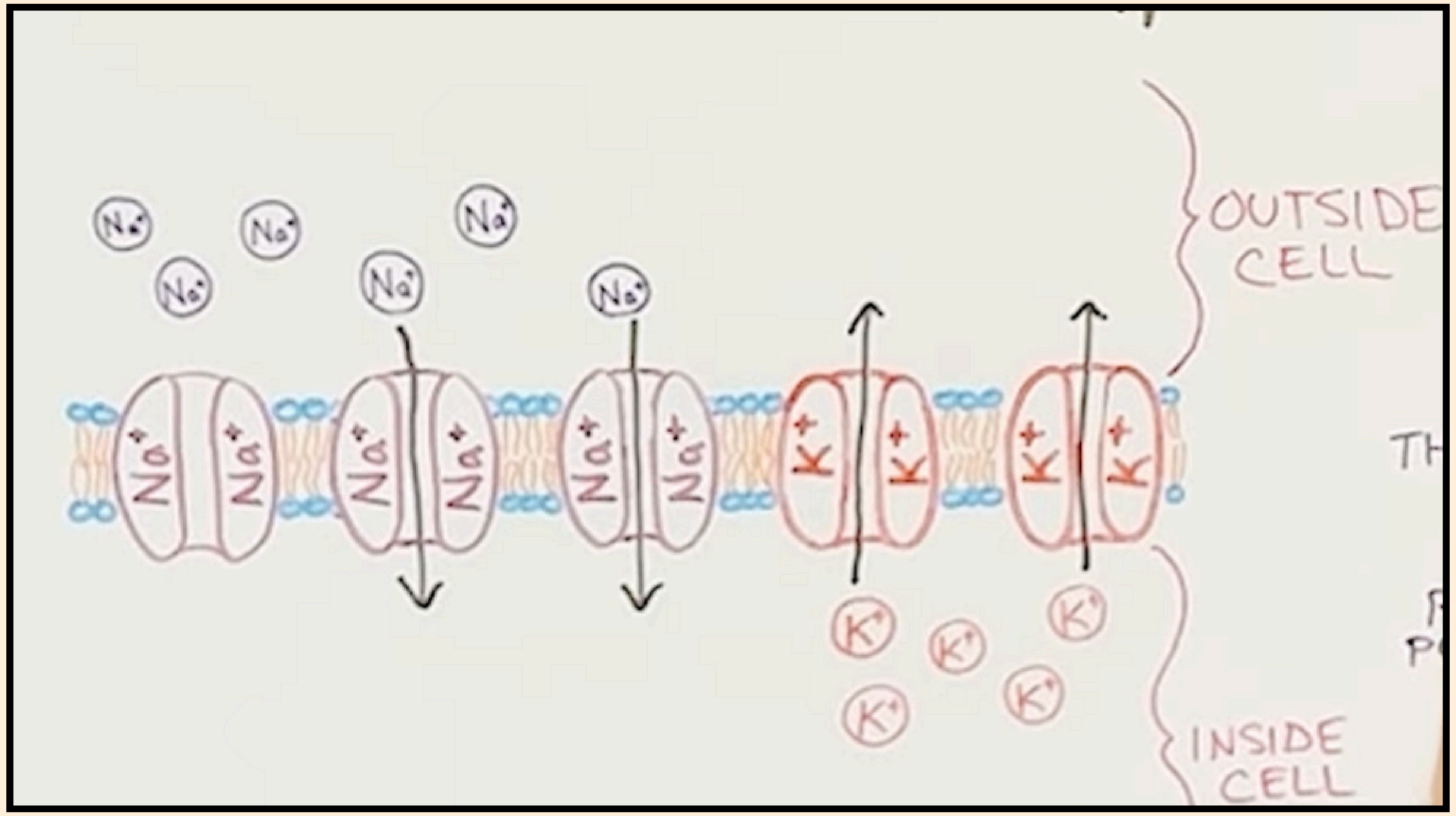


AHP Type Current



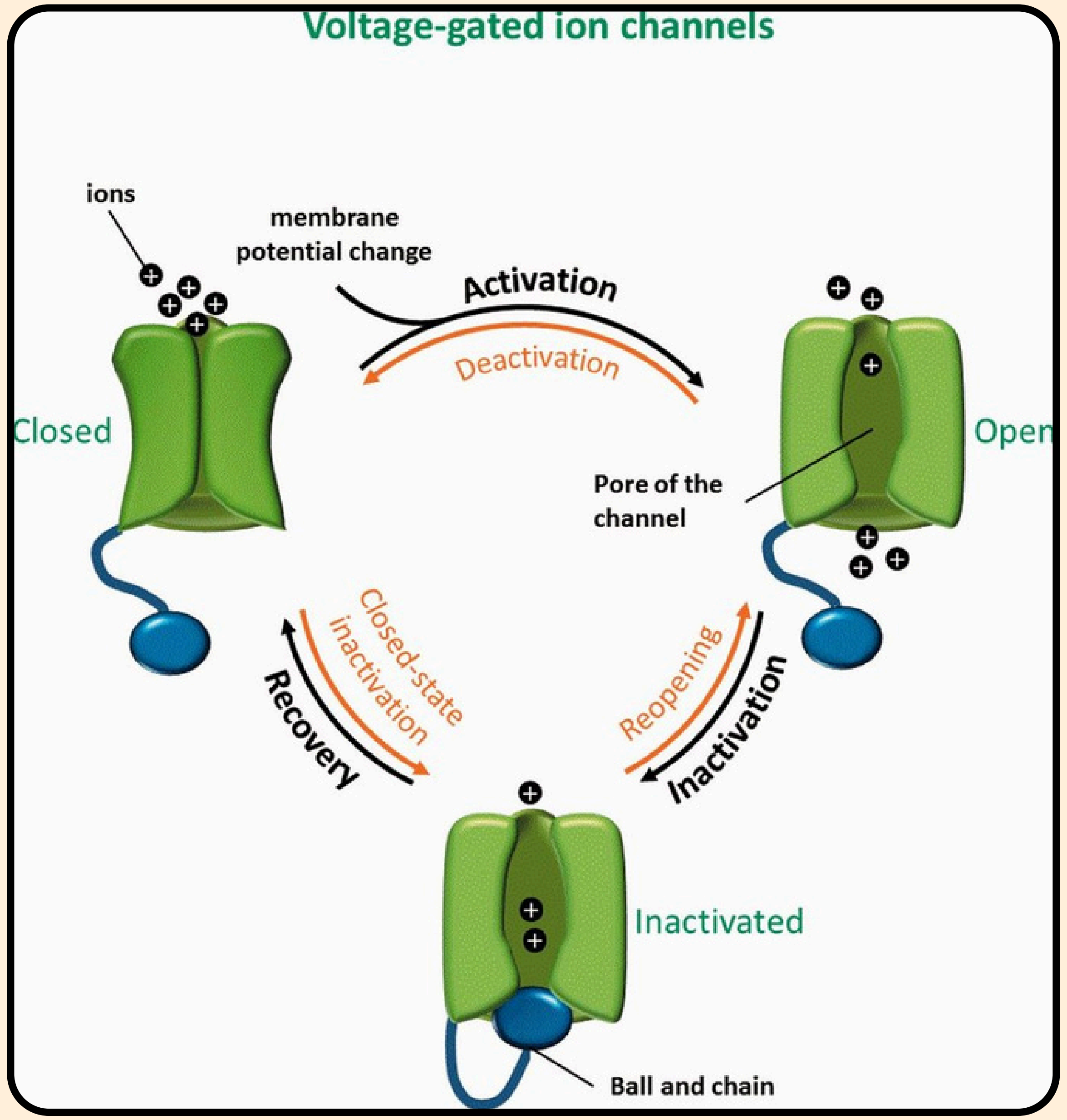
AHP TYPE CURRENT

- AfterHyperPolarization (AHP) current co mediated by the calcium-dependent activation of potassium (K+) channels rather than voltage alone. Each action potential triggers a transient influx of calcium ions (Ca2+) through voltage-gated channels
- As this calcium accumulates in the cell with repetitive spiking. This rising calcium concentration progressively activates K-Ca channels, generating a sustained outward potassium current that hyperpolarizes the membrane and increases the refractory period between spikes.



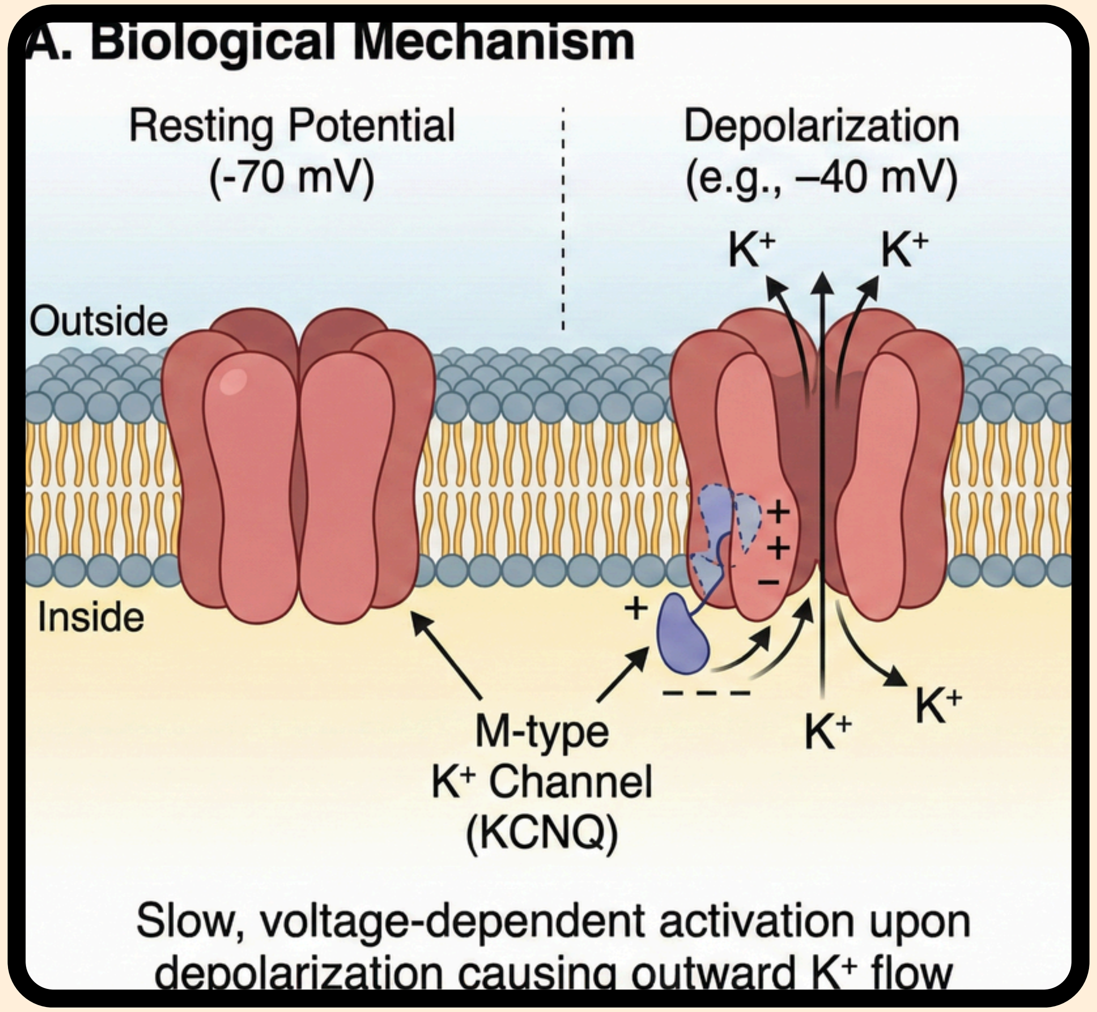
SLOW RECOVERY OF FAST SODIUM (NA+) CHANNELS

- While the standard action potential cycle involves a rapid transition from open to "fast-inactivated" states– defining the absolute refractory period- repetitive sustained depolarization drives a distinct fraction of these channels into a deeper, "slowly inactivated" conformational state.
- Unlike the millisecond-scale recovery of standard inactivation, this slow state requires a prolonged duration of membrane hyperpolarization to reset the channels to a "closed-but-ready" status.
- the density of available Na+ channels decreases, effectively raising the firing threshold and often attenuating the amplitude of subsequent spikes.



IM CURRENT

- M-type current (IM) mediated by slowly activating, non-inactivating voltage-gated potassium channels. It activates in the subthreshold-to-near-threshold range and persists throughout sustained depolarization without closing.
- This process creates a negative feedback loop where the accumulation of depolarization makes the neuron progressively "leakier" to current, thereby dampening excitability.



RC CIRCUIT ADAPTATION OF LIF MODEL

ADDING A SLOW-DISCHARGING RC TO REDUCE THE FIRING RATE

- An additional RC network (Radapt and Cadapt) is connected to the output. It acts as an integrator that tracks recent activity.
- Every time the neuron spikes, a small amount of charge is dumped onto Cadapt. Radapt is chosen to be very large to increase the time constant of the network (Radapt*Cadapt). This prevents voltage from increasing instantly after a spike.
- The voltage across Cadapt drives a feedback transistor that pulls current away from the main membrane node
- As the neuron fires repeatedly, the feedback voltage builds up -> inhibition increases -> net charging current decreases -> inter-spike interval increases.

CIRCUIT DYNAMICS & GOVERNING EQUATIONS

The Time Constant: The adaptation timescale is determined by the RC product. It must be significantly slower than the membrane time constant to span multiple spikes.

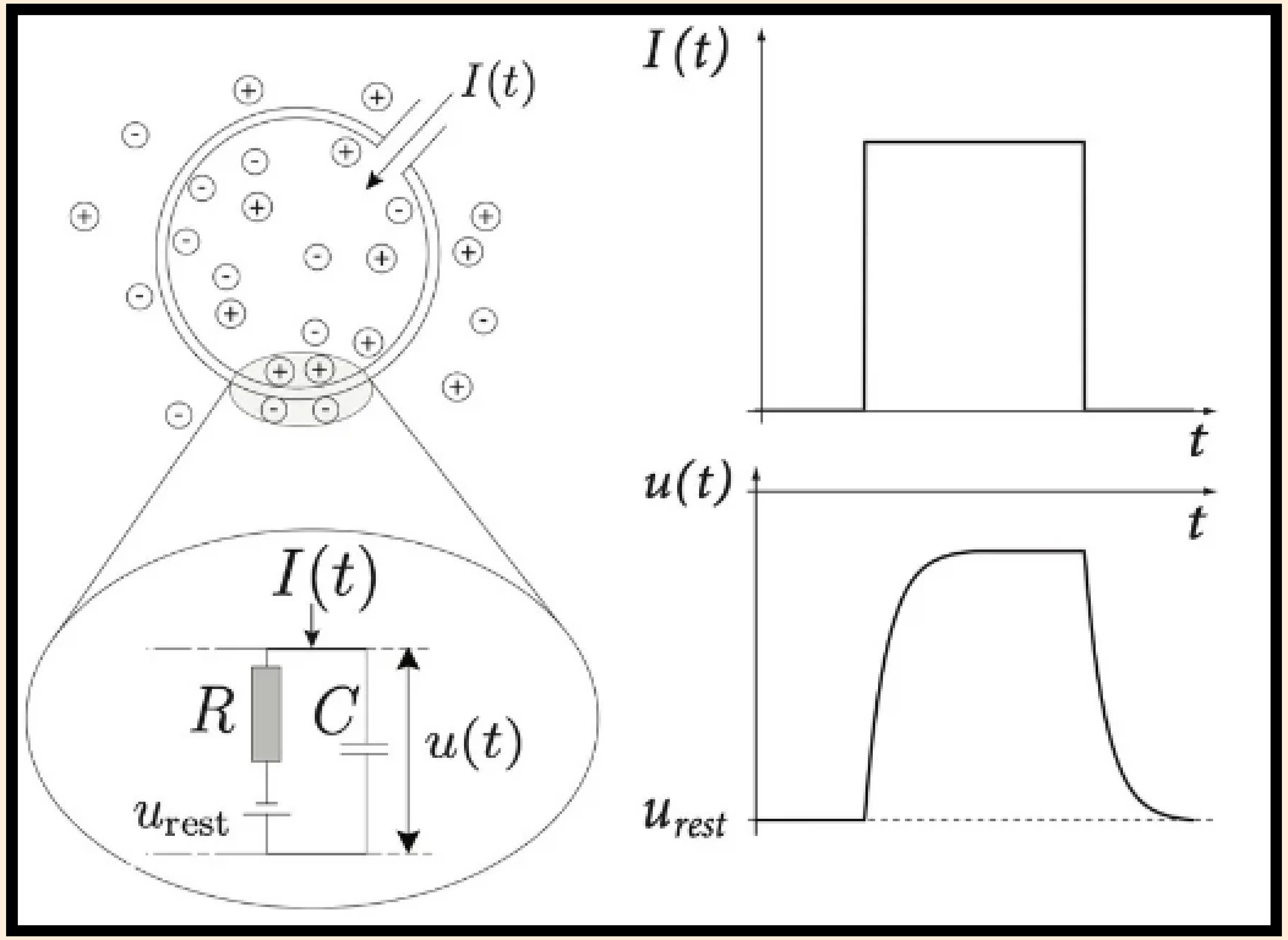
$$\tau_{adapt} = R_{adapt} \times C_{adapt}$$

Adaptation Variable Dynamics: The voltage/current responsible for adaptation (ω) increases by a step (b) at every spike time (t_f) and decays exponentially between spikes.

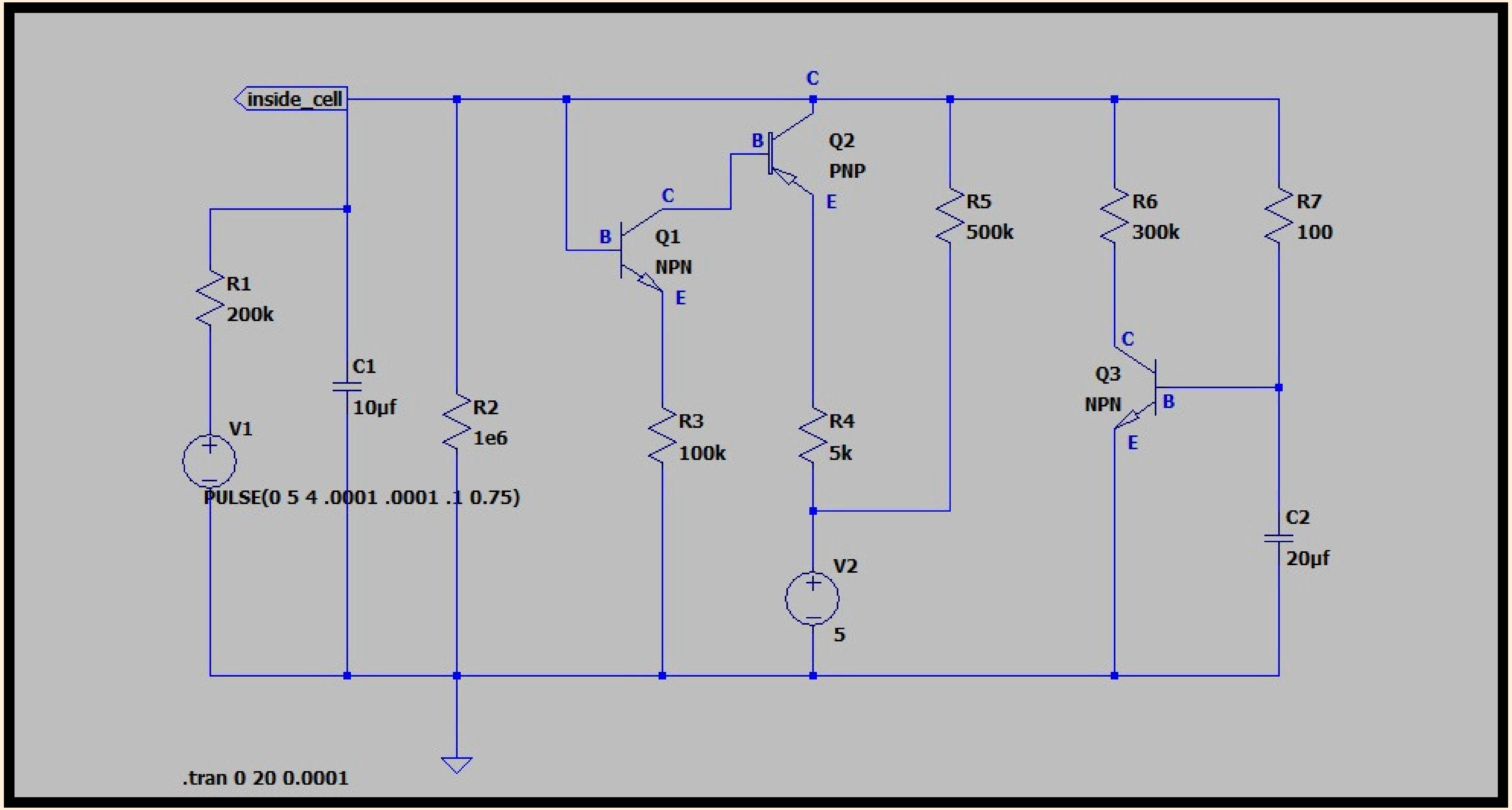
Effective Input Current: The adaptation variable acts as a subtractive term from the input stimulus ($i_{stimulus}$), effectively slowing down the membrane charging rate dV/dt.

$$C_{mem}\frac{dV}{dt} = I_{stimulus} - \frac{V}{R_{leak}} - w$$

CIRCUIT SIMULATION



BASIC LIF CORE CIRUIT



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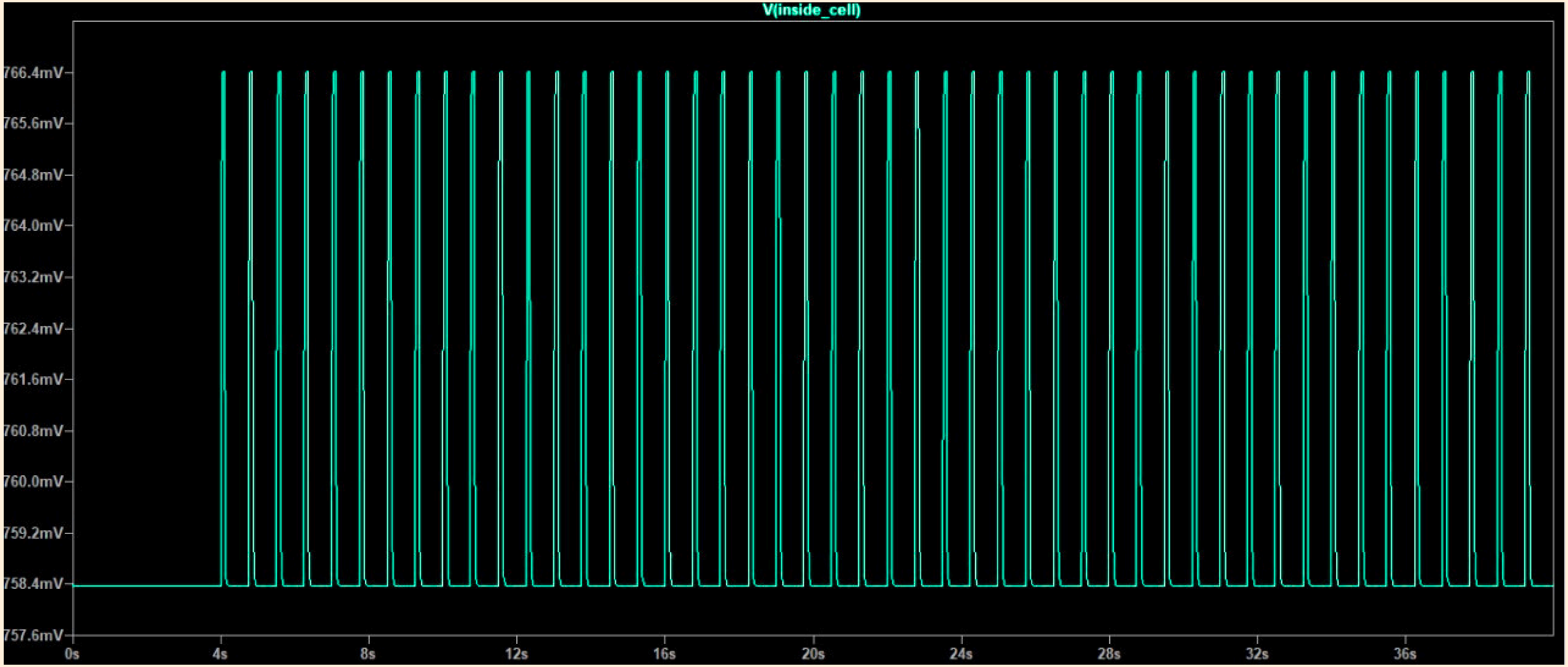


EFFECT OF VARIOUS DISEASES

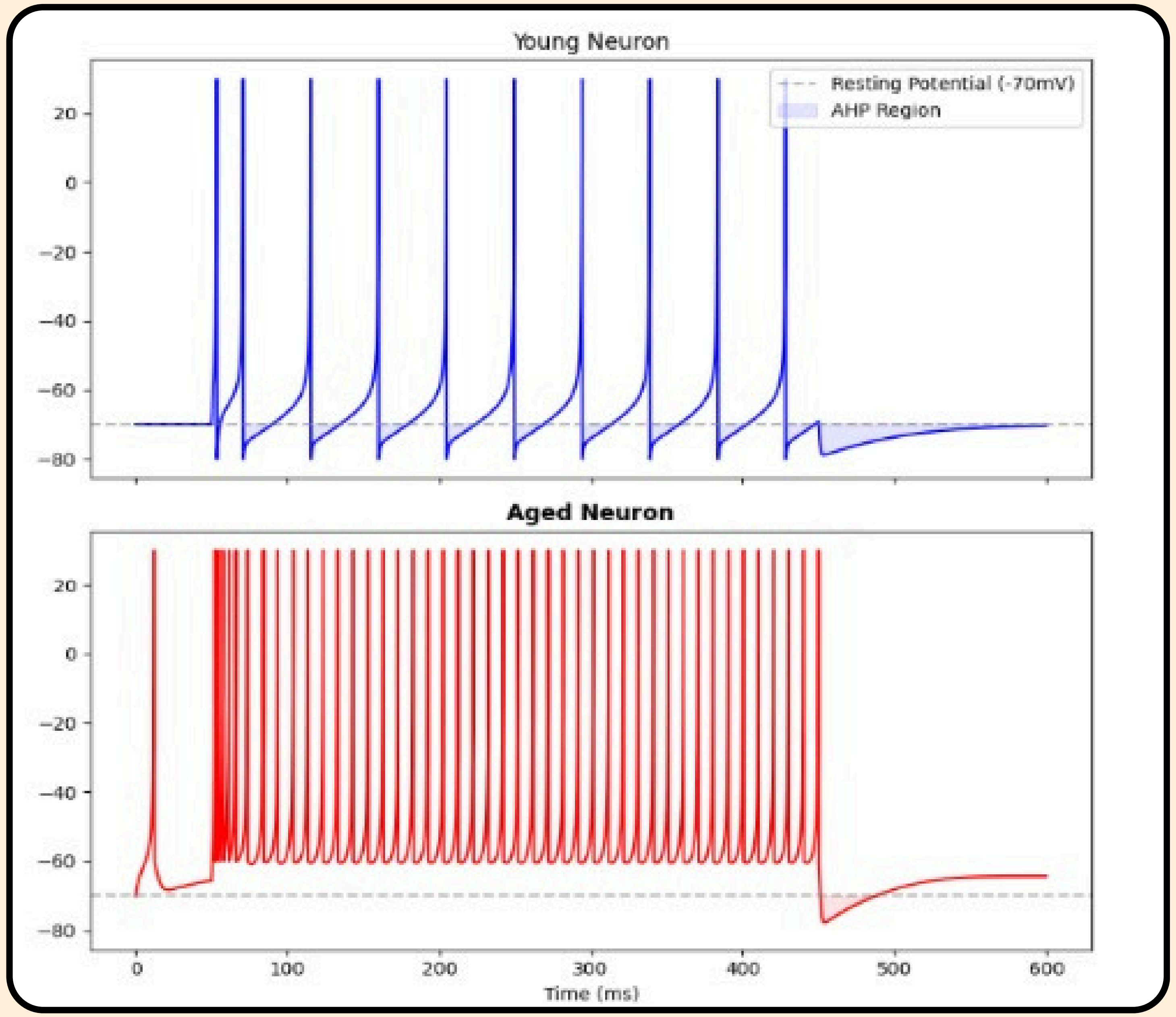
COMPARATIVE ANALYSIS: HEALTHY YOUNG VS. PHYSIOLOGICALLY AGED

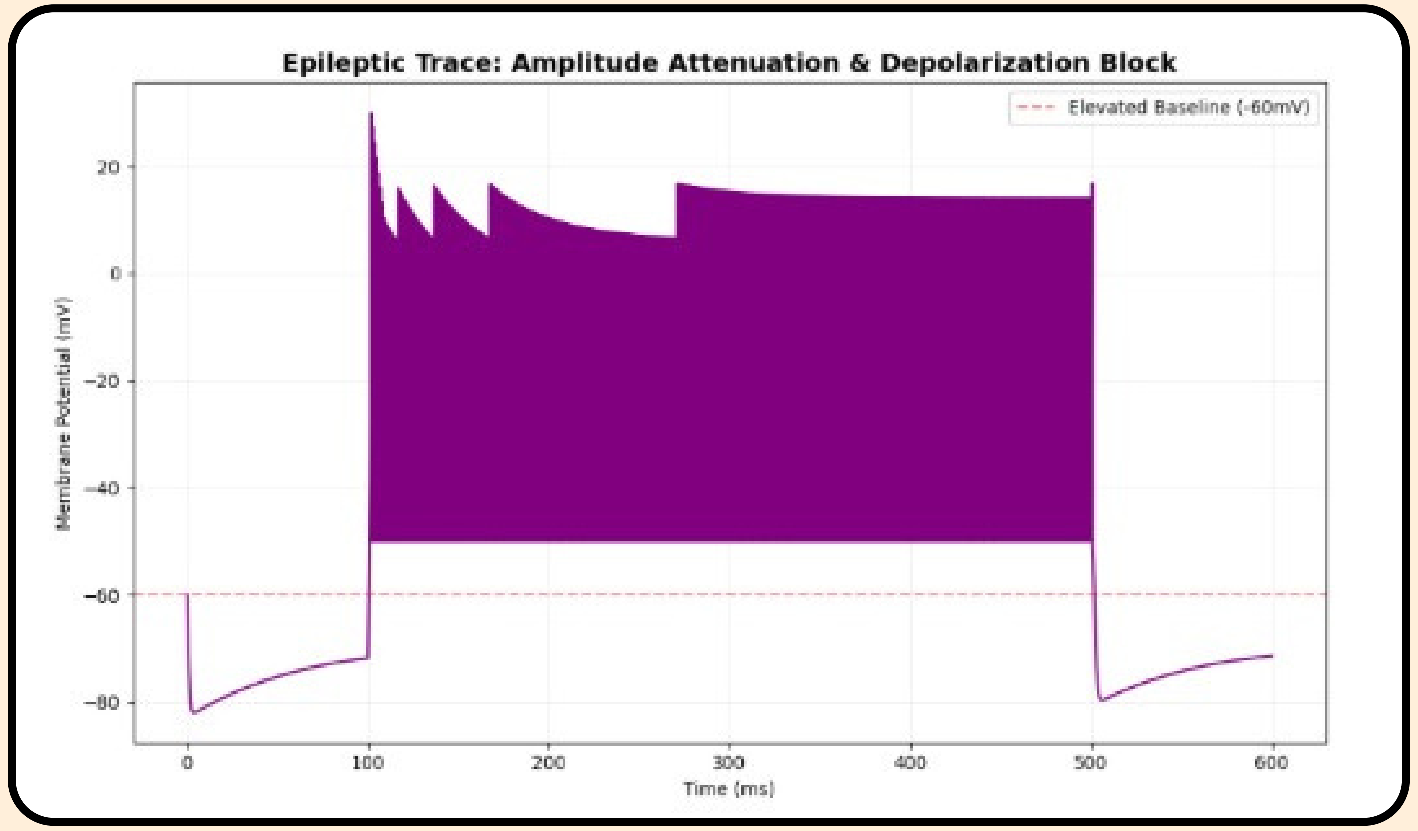
- Signal Amplitude: While healthy young neurons generate high-amplitude spikes with significant overshoot, aged neurons display attenuated amplitude caused by downregulated sodium channel density.
- Firing Pattern: Young neurons demonstrate strong logarithmic spike frequency adaptation, whereas aged neurons exhibit a linear, "metronomic" firing pattern due to impaired calcium-SK channel coupling.
- Reset Mechanism: The deep after-hyperpolarization (AHP) characteristic of youth is replaced by a shallow reset phase in aging, rendering senescent neurons highly susceptible to immediate re-excitation.

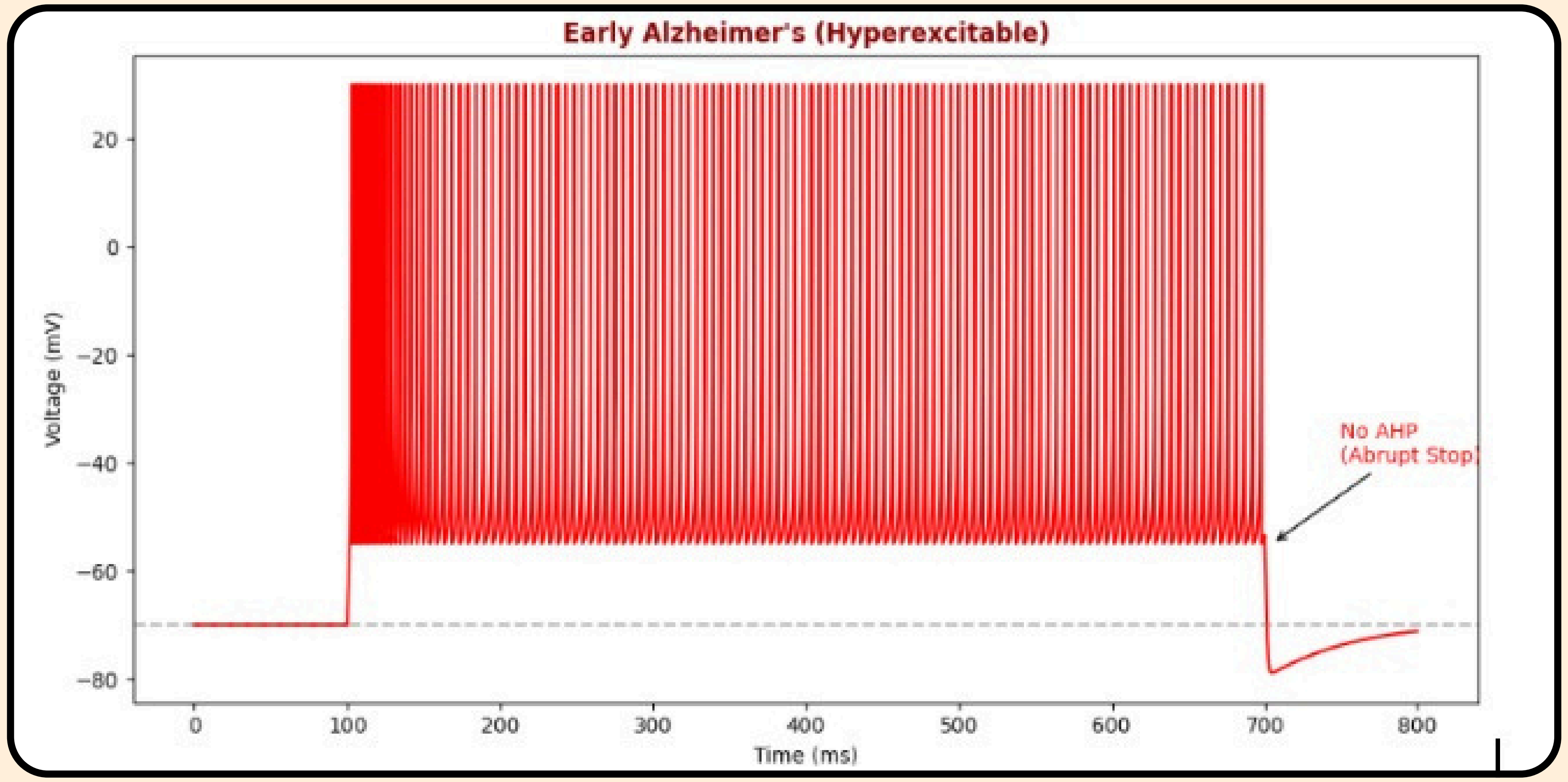
Pathological States: Early Alzheimer's & Epilepsy

- Alzheimer's (The Panic Mode): Soluble Amyloid-β triggers the internalization of SK channels, destroying the neuron's braking mechanism and causing a shift from adaptive coding to tonic, panic-like firing.
- Epilepsy (The Crash): The failure of the stabilizing M-Current allows runaway excitation to overwhelm sodium channel recovery, forcing the neuron into a "depolarization block" where spiking physically ceases at a high-voltage plateau.
- Quantitative Progression: The severity of dysregulation is quantifiable by firing rate, escalating from a controlled 23.33 Hz in healthy tissue to 198.33 Hz in Alzheimer's, and finally reaching a chaotic 455.00 Hz in the epileptic state.

ANALYSIS


Young Neuron
Resting Potential (-70mV)
AHP Region
Aged Neuron
Time (ms)


Epileptic Trace: Amplitude Attenuation & Depolarization Block
Elevated Baseline (-60mV)
Membrane Potential (mV)
Time (ms)


Early Alzheimer's (Hyperexcitable)
No AHP
(Abrupt Stop)
Voltage (mV)
Time (ms)

Summary Metrics

MODEL	FIRING RATE (Hz)
Young	23.33
Aged	106.67
Alzheimer	198.33
Epilepsy	455.00

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CITATION

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