Section 2-1
Cells

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COGS 17 A04

01/24/25

Notice

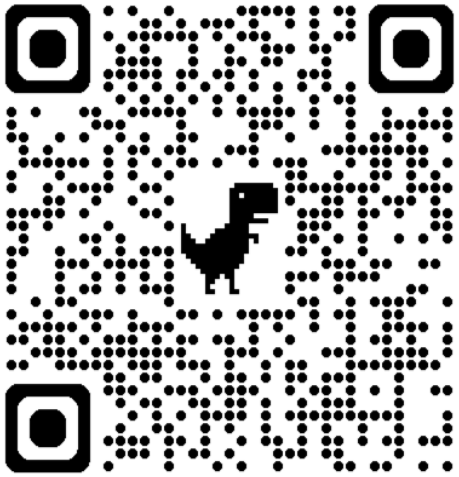
MIDTERM I (125 Points)– Next Tue!

3:30-4:50 pm (80 minute)

No Class - Exam Online

Neuroanatomy, Cells, Development

For Section Slides: :



Activity for this week's section

Crossword

Words can go across or down.

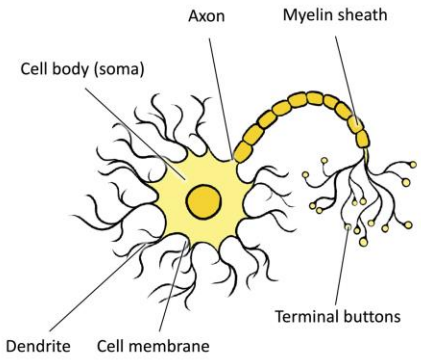
Common features of Cells

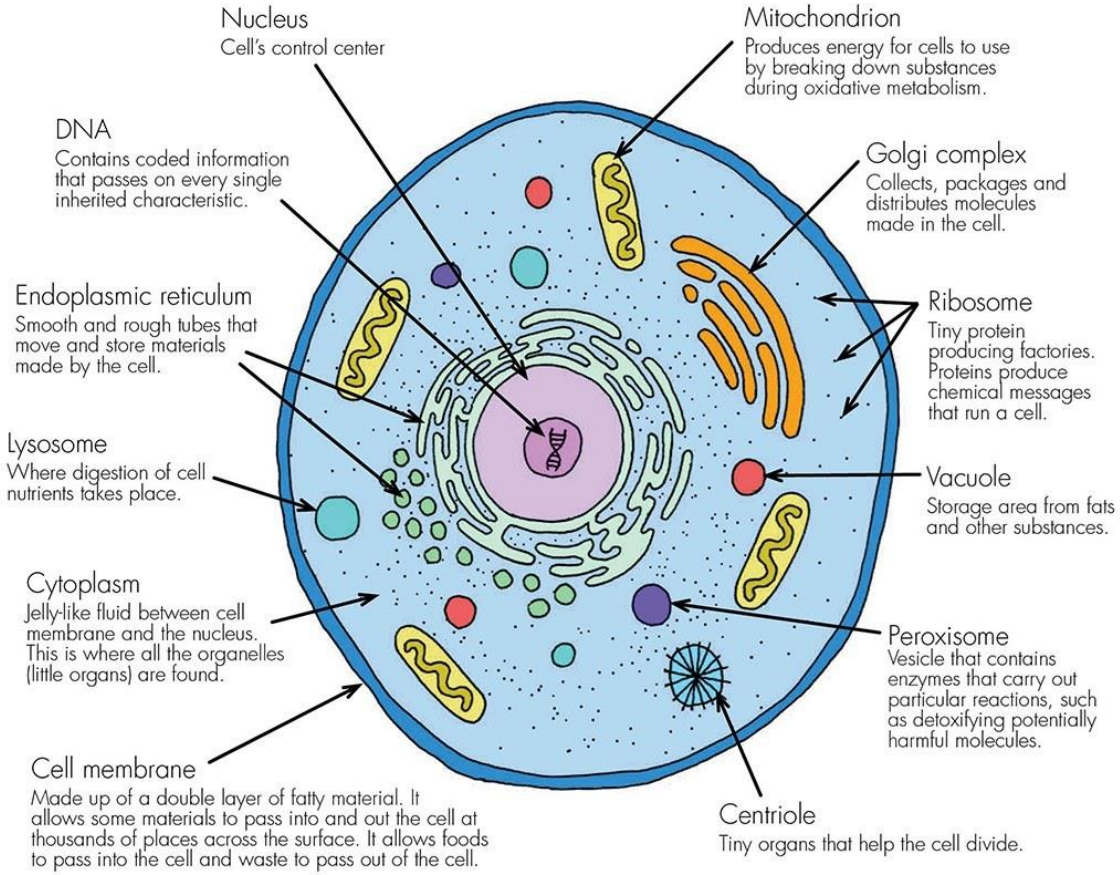
- Soma
  Fancy word meaning “cell body”

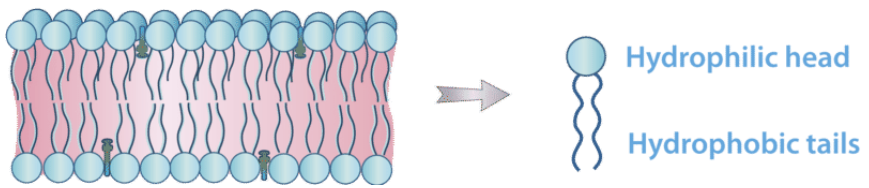
- Cytoplasm
  Fluid within a cell

- Extracellular Fluid
  Fluid outside of a cell

- Cell Membrane
  A double layered wall consisting of lipids (fat molecules)





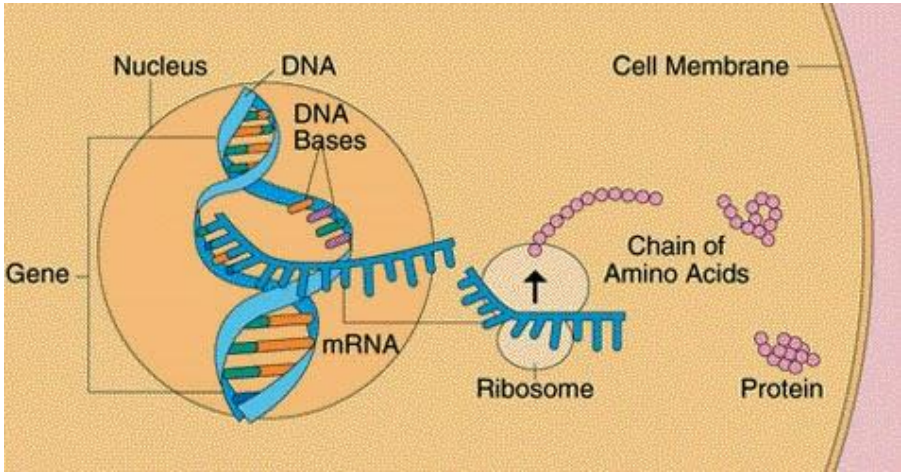


Important Organelles to Remember

- Nucleus
  An inner “control center” where **DNA** is stored

- Ribosomes
  Small protein producing factories

- Mitochondria
  The “powerhouse of the cell”



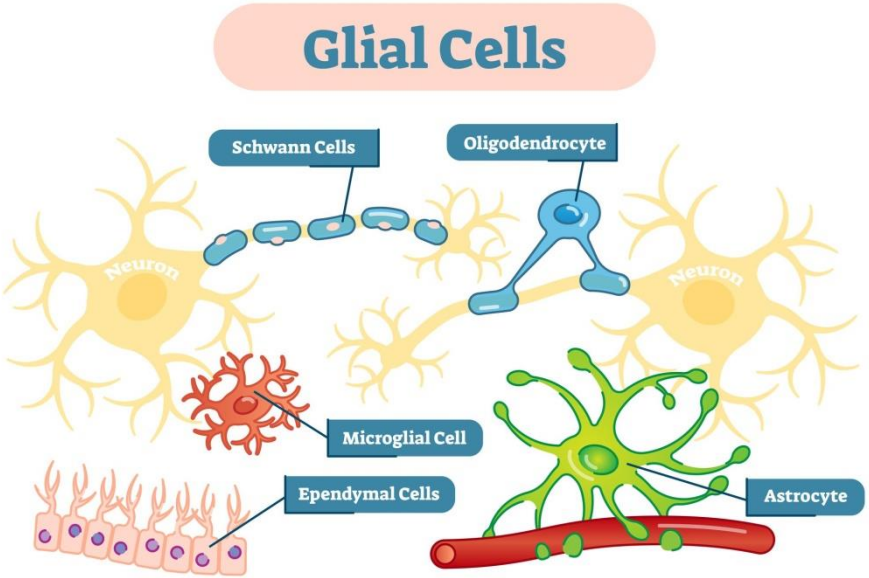
Specialized Cells of the Nervous System

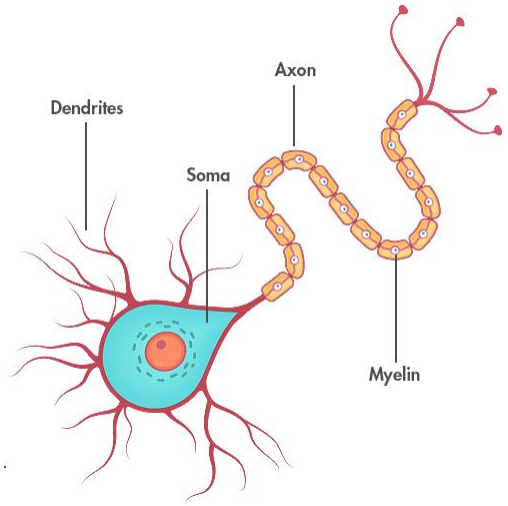
- Neurons
  Cells that are specialized for Information Transfer via **Processes** and **Membrane**

- Glia Cells
  Have many functions but do not participate in Information Transfer

  "Glia" meaning "Glue" which holds the nervous system together, both physically and chemically, to support Neurons

  1/10 size of a neuron, x10 times as many, 50% of brain by weight





Different Glia Cells

- **Radial Glia**

Guide the migration and growth of neurons during fetal development

- **Ependymal Cells:**

Lines ventricles and act as a layer between the ventricular cavities and the parenchyma

Secretes CSF into the Ventricles

- **Oligodendrocytes**

Surrounds axons in a process called myelination in the CNS

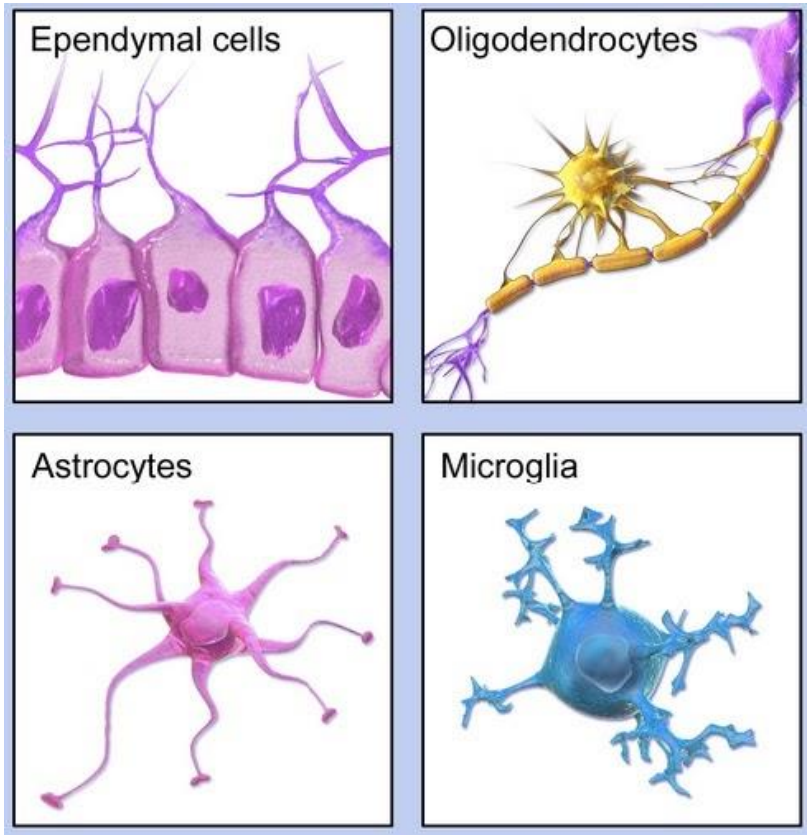
Schwann Cells: specialized Oligos which myelinate neurons of the PNS

- **Astrocytes**

Provides nutrients, recycles NTs, maintains the BBB, and numerous other functions

- **Microglia**

Removes toxins from the brain, repairs damaged neurons



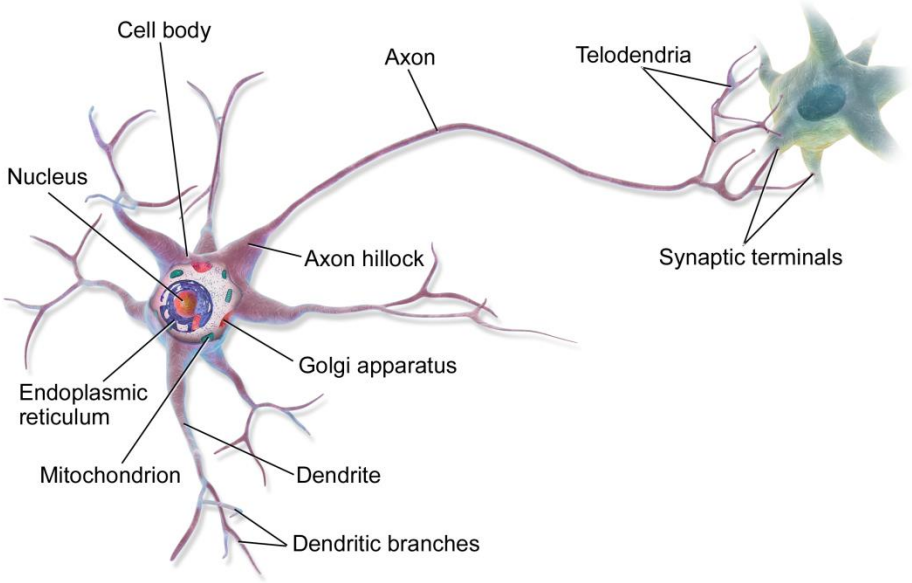
Neurons

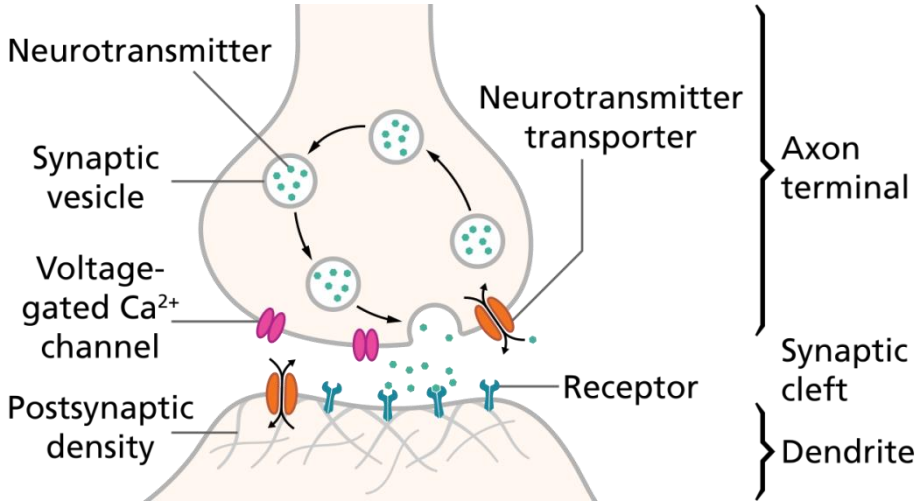
- Specialized cells for information transfer

- <u>Dendrites:</u>
  - Spiny protrusions from the Soma which receives **incoming** signals
  - Site of Postsynaptic Membranes

- <u>Axons</u>
  - Long fibers which reach out to other neurons
  - Carries **outgoing** signals
  - Terminates in Presynaptic Terminals (a.k.a Terminal Buttons, or End Bulbs) which releases NTs into the Synaptic Cleft

- <u>Receptor Sites:</u>
  Specialized areas which interact with NTs from other neurons





Important Concepts

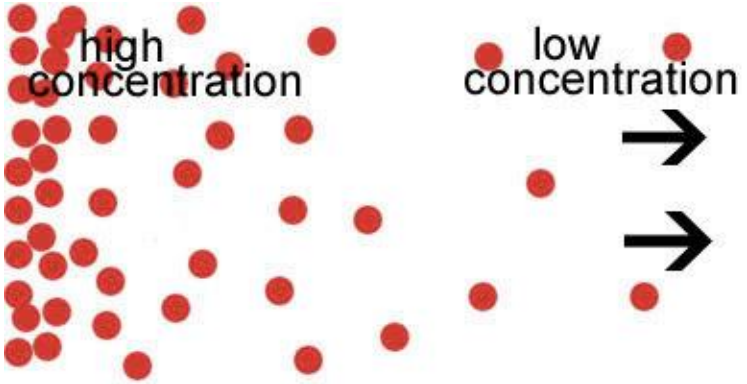
- Nature always seeks **Equilibrium**

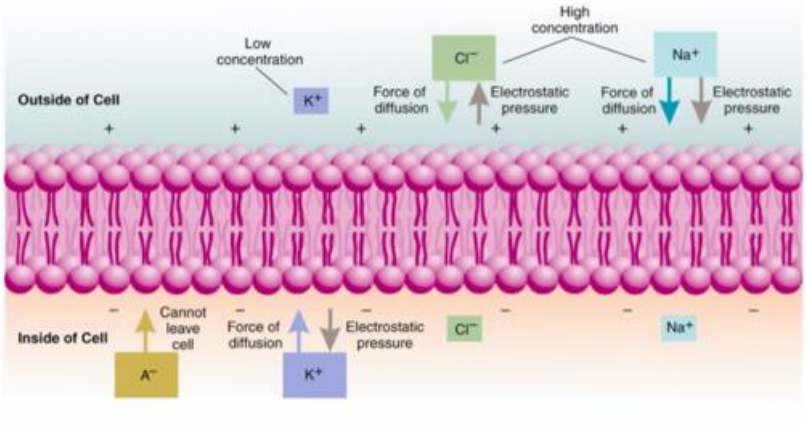
- Concentration Gradient:
  - Molecules in areas of **greater** concentration will diffuse to areas of **lesser** concentration
- Electrical Gradient:
  - Negative repels negative charges (heading towards positive charges) and positive repels positive charges (heading towards negative charges)

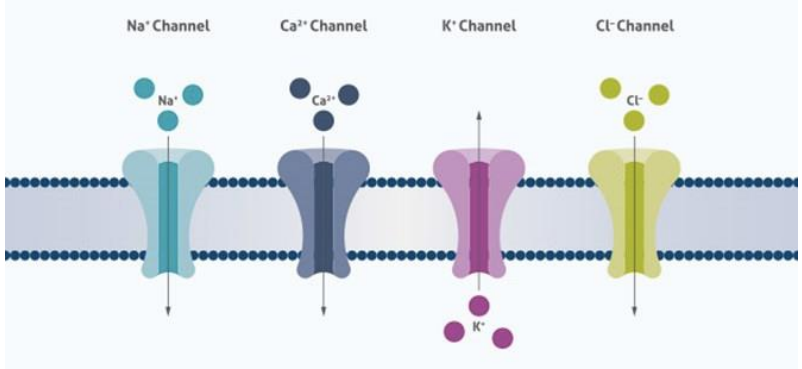
    = **Electrostatic Pressure**

- Selective Permeability of Membranes
  - Lipid bi-layers are typically impermeable to charged ions and large molecules
  - Selective Permeability allows membranes to control which chemicals enter/leave the cell, affecting the electro-chemistry
  - Important Ions to remember:

    Na^+, K^+, Ca^{2+}, Cl^-







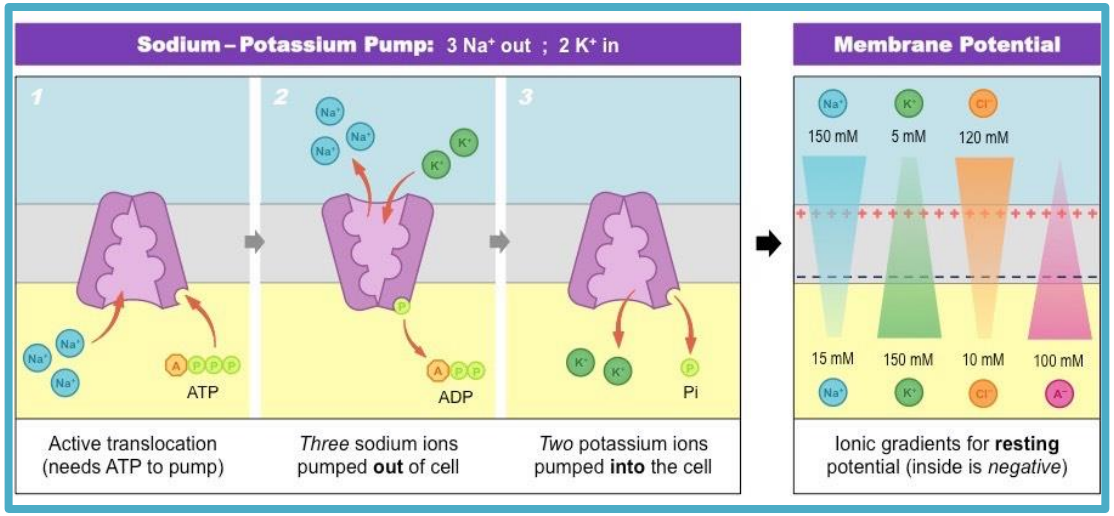
Resting Potential

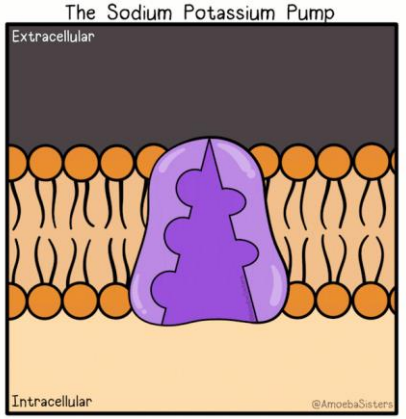
- Membrane Potential
  - The difference in charge between the inside and outside of the cell. Measured in milli-volts

- Resting Potential:
  - typically **-70 mV** for Neurons (more positive outside)

- Sodium/Potassium Pump
  - Helps establish resting potential by transporting **3 Na^+ out** and **2 K^+** ions **in**
  - Na^+ concentration ratio is 10:1 (out:in) which means **Na^+** ions **want to enter the cell**
  - K^+ concentration is 1:10 so it wants to **exit** the cell, but is **prevented** by the electrical gradient

  - Closed Ca^{2+} gates keep Ca^{2+} out of the cell + electrical gradient keeps Cl^- out
  - Resting Neurons are **Polarized (different charge in/out)**





Action Potential (AP)

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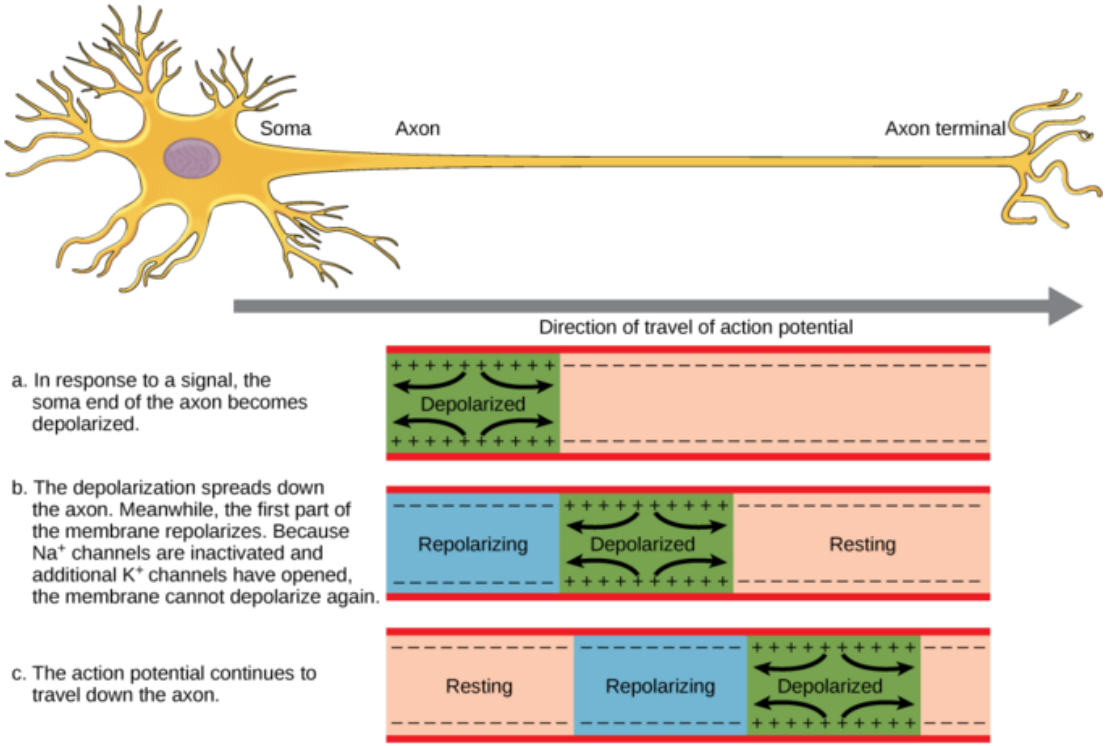
- Depolarization of the Neuron
  - If Resting Neurons are **Polarized**, then **Depolarized** neurons are not “resting” AKA neurons are “firing”

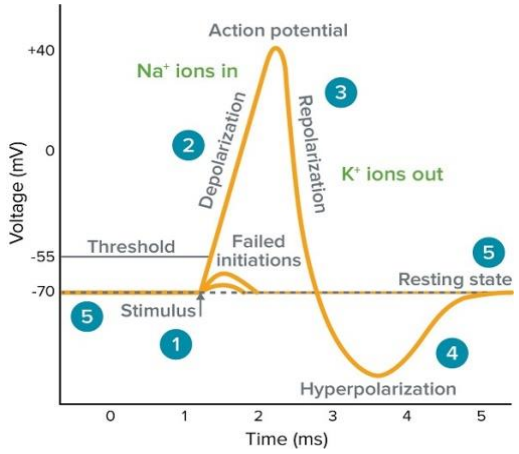
- Propagation process
  - Stimulation from Presynaptic neuron → release of NTs → binds to Postsynaptic neurons **>** triggers AP that starts at the Axon Hillock



Action Potential (AP)

- Mechanism of Action
  - Na channels open, allowing an **influx** of **Na** ions, drastically shifting the membrane potential towards a peak of **+50** mV
  - At the peak, Na channels close while K channels open, allowing an **efflux** of **K** ions, shifting the membrane potential negatively to a point where it overshoots (hyperpolarizes)
  - K channels close and **Na/K pumps** start re-establishing resting potential (via 3 Na out, 2 K in) until membrane potential returns to -70 mV, This time period is called **Refractory Period**, during which the neuron cannot fire
  - Calcium pumps at the Axon Terminal actively transports Ca out to reset the NT release mechanism

- All or None Law
  - In a given cell, AP will always have the same amplitude and velocity regardless of the intensity of the stimulus that triggered it

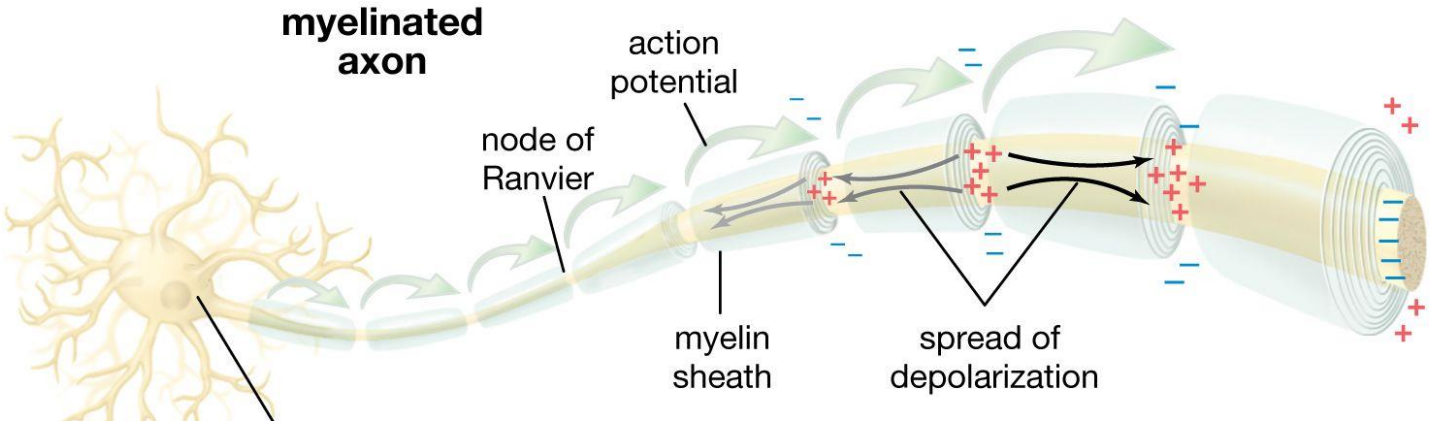


Myelination

- Speed up AP

- Glia cells wrapped around the axon act as an insulator
  - Oligodendrocytes in CNS and Schwann cells in PNS
- **Electrical conduction** in myelinated portions

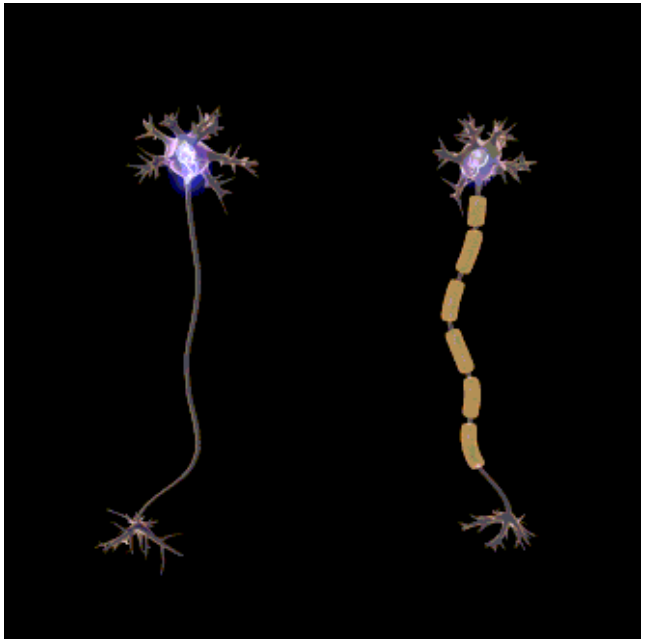
- <u>Nodes of Ranvier:</u>
  - The small gaps between myelin sheaths
  - sustain **Ionic Conduction** (when charged atoms flow through pores in the cell membrane) boosts the electrical signal

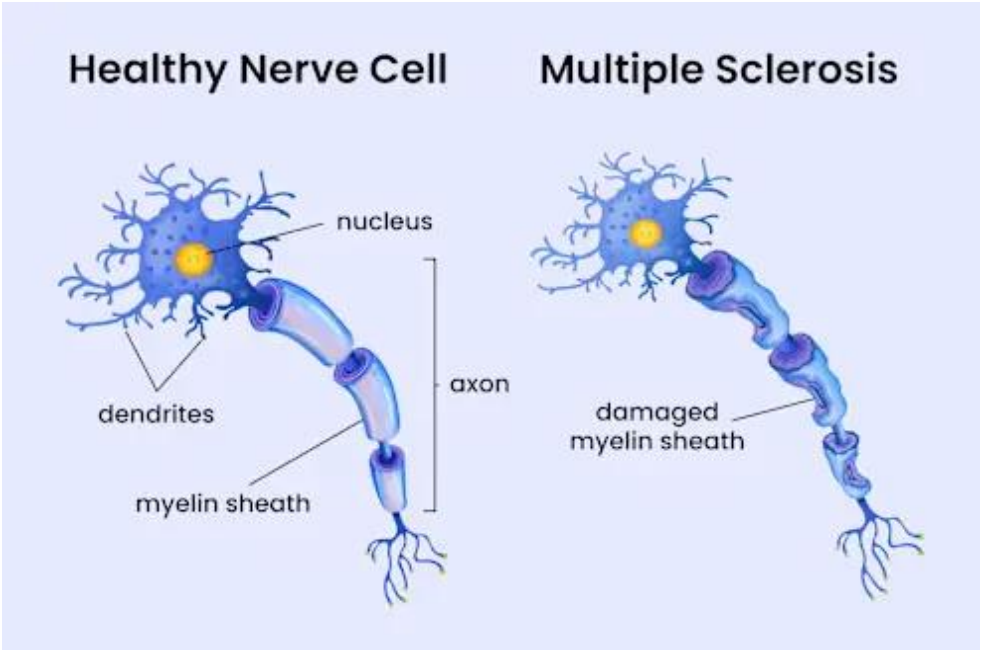


Myelination

- <u>Saltatory Conduction:</u>
  - Nerve impulse “jumps” from one node to another in a myelinated cell
  - Increases overall speed of impulse

- <u>Multiple Sclerosis (MS):</u>
  - A neurodegenerative disease where myelin degrades over time
  - Electrical signals decay quickly and AP fail





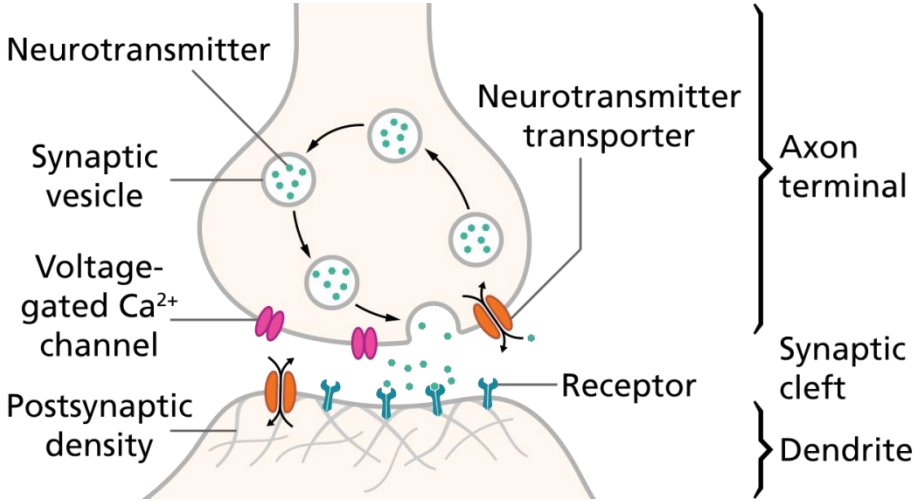
The Synapse

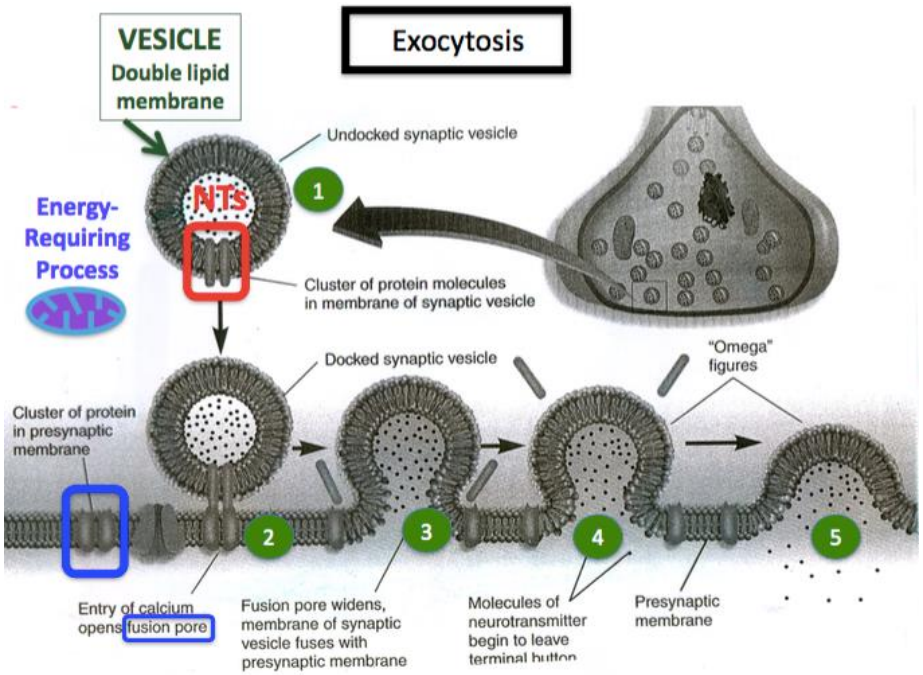
- Presynaptic cell + Synaptic Cleft + Postsynaptic cell = The Synapse

- Presynaptic cells release NTs into the cleft via **Exocytosis**
  - NTs are packaged into vesicles

- **Influx of Ca** initiates the exocytosis
  - Ca opens the Fusion Pore which **binds vesicles** to the presynaptic cellular membrane

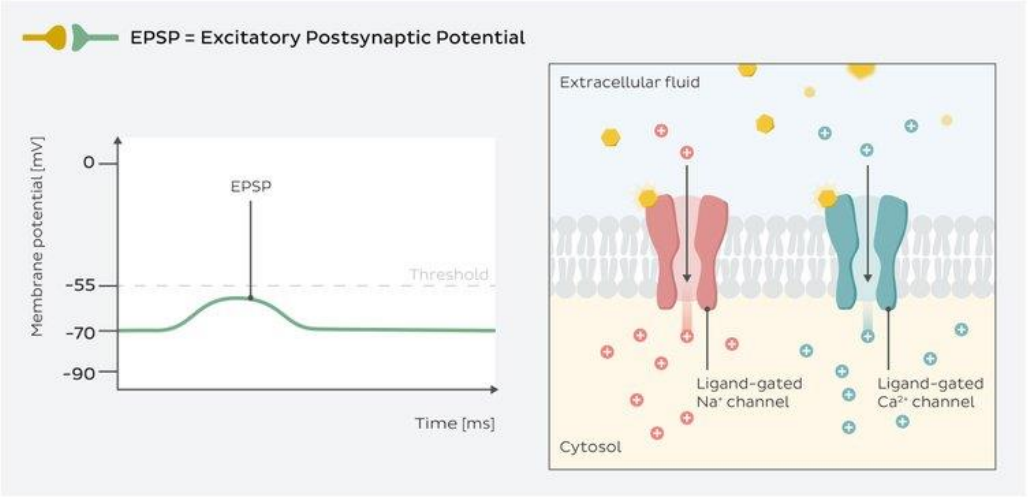
- Following exocytosis, **NTs** passively **diffuse** across the synaptic cleft and binds to NT-specific receptor sites on postsynaptic neurons

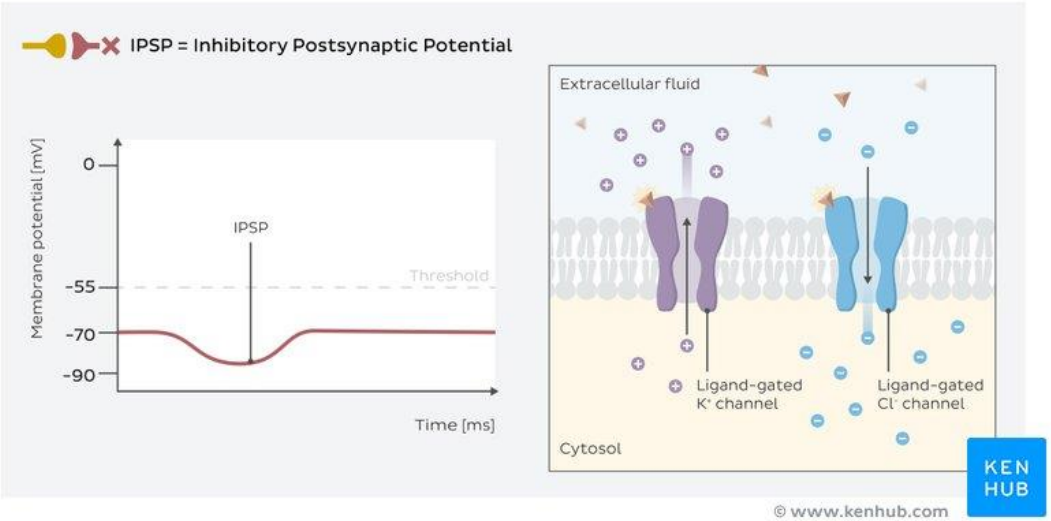




Polarity of Postsynaptic Cells

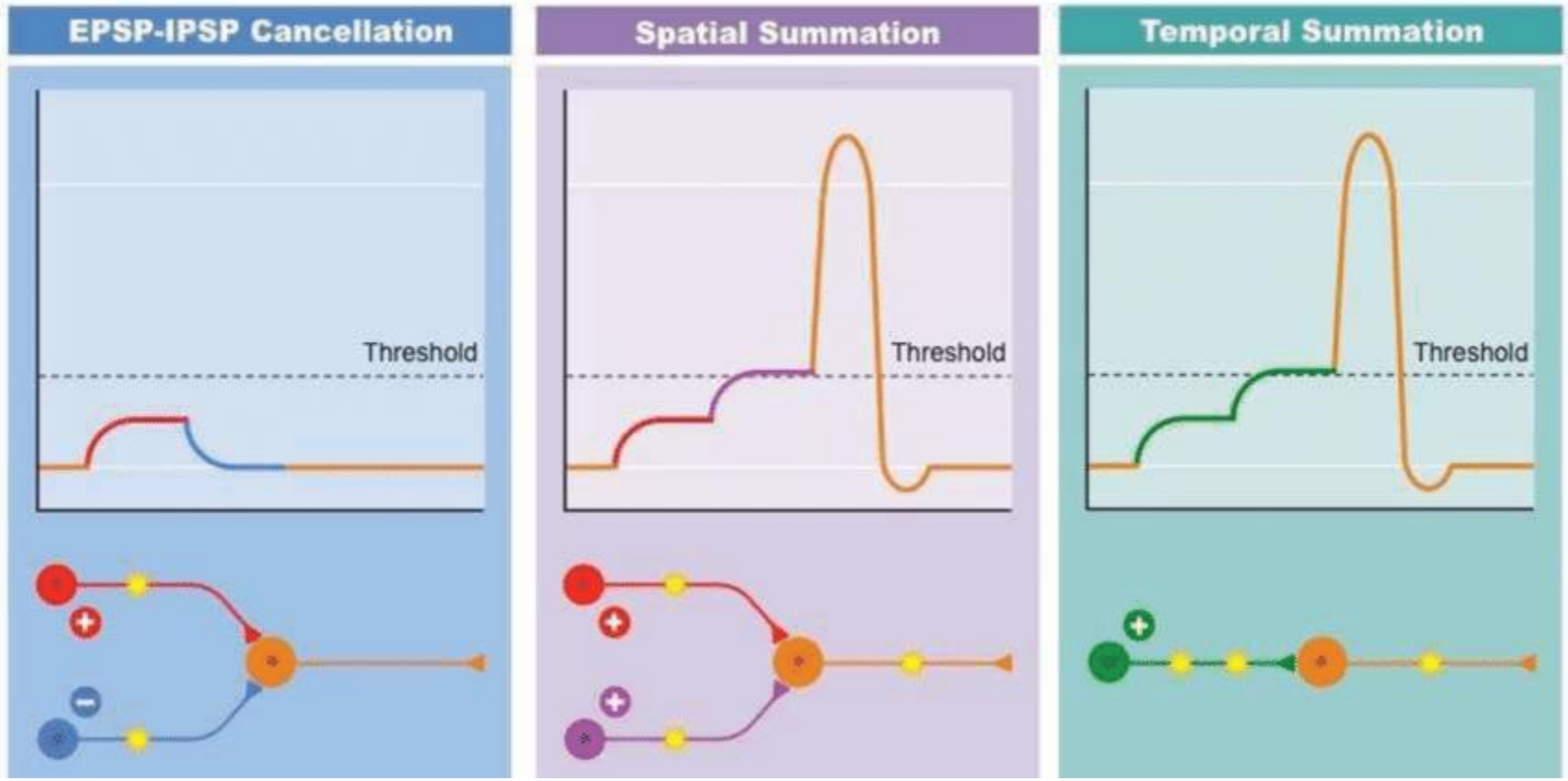
- **EPSP**
  - Increases a cell's likelihood of releasing NTs, **more** likely to "fire"
  - Usually due to Na+ entering the cell
- **IPSP**
  - Decreases a cell's likelihood of releasing NTs, **less** likely to "fire"
  - Usually due to K+ entering or Cl- exiting



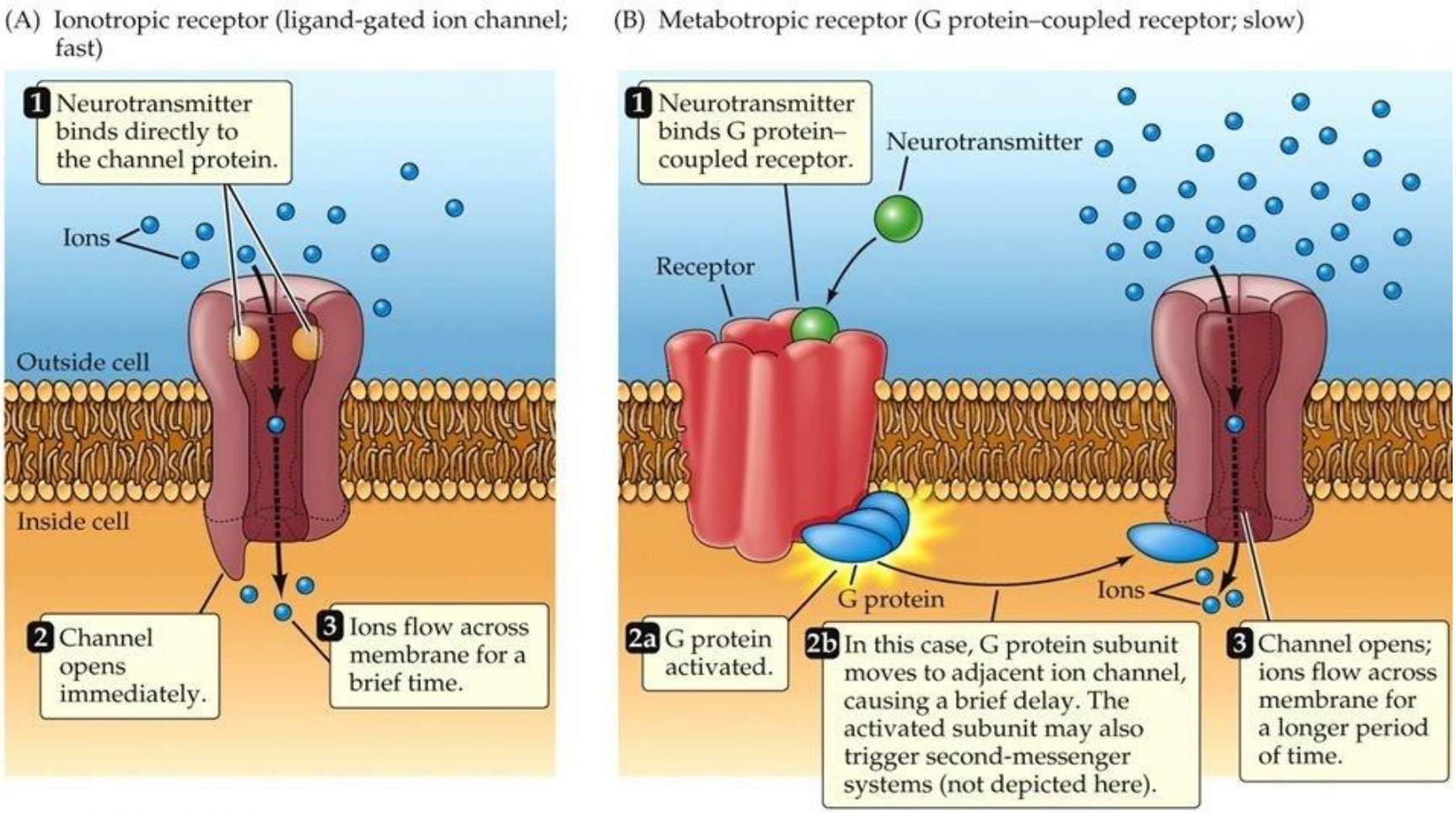


Polarity of Postsynaptic Cells

- Summation
  - A neuron's response = sum of EPSPs and IPSPs
  - **Temporal Summation:** one or more cells **repeatedly** stimulate another in rapid succession
  - **Spatial Summation:** multiple cells converge on a single **location** on a cell at the same time



Mechanisms of Neurotransmitters



Ionotropic

- Directly affects ion gates
- Rapid and Short-lived responses
- Best for sending info about changing inputs

Metabotropic

- Causes metabolic changes in Postsynaptic cell
- Activation of G protein and second messenger
- Slower but long-lasting responses

Some Neurotransmitters and their Functions

Chemicals are called NTs if they impact nearby neurons

Neurotransmitter	Functions
Acetycholine (Ach)	• All neuro-muscular junctions • Cortical arousal
GABA	• Most common inhibitory NT • Regulate anxiety
Glutamate	• Most common excitatory NT • Learning • Perception • Schizophrenia
Serotonin (5HT)	• Often acts as a neuromodulator • Mood regulation, sleep, perception
Dopamine	• Reinforcement • Attention • Motor control
Norepinephrine	• Arousal • Attention
Epinephrine (adrenalin)	• Arousal • Attention
Substance P	• Pain (damage, itch, extreme temperatures, etc)
Endorphins	• Counter effects of Substance P
Hormones	• Testosterone, estrogen, cortisol, oxytocin, endorphins, etc

Agonist vs Antagonist

- **Agonists**: **Increases** the effect of a NT
- **Antagonist**: **Decreases** the effect of a NT

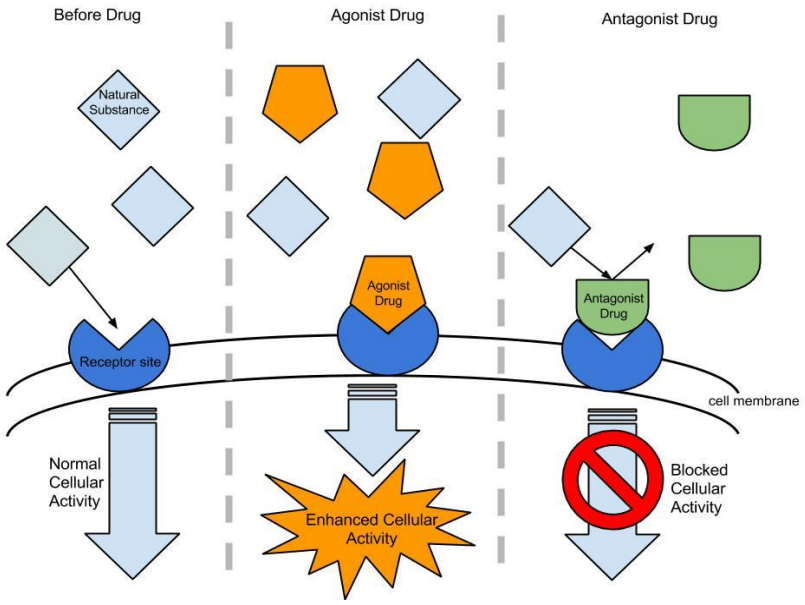
Some examples:

- Acetylcholinesterase
  - Enzyme which breaks down Ach in the cleft

- Serotonin Reuptake
  - Prosac (antidepressant): serotonin reuptake inhibitor (SSRI), increasing the NT's duration in the cleft

Agonists and Antagonists can also act inside the presynaptic cell to affect NT release:

- Some antagonistic drugs (e.g. Reserpine) prevent NTs (Monoamines) from being packaged into vesicles
- Some agonists (like Black Widow Spider venom) cause massive release of NT (ACh)



Other Factors affecting Function

1. Activation of DNA sequences initiated the production of proteins for structural and chemical changes within cell
2. Repeated activity leads to more dendritic spines and more receptor sites (# of receptor sites)
3. Receptor Sites can be blocked by NT mimics that do not readily detach
   - E.g., LSD binds to Serotonin sites
4. Some NTs may require Hours/Days to replenish
   - Carried by Kinesin molecules (walk along micro-tubules from soma to terminal)
5. Some NT precursors can pass the BBB and be used as medication (e.g., L-DOPA)

<u>Exceptions: Receptor Sites on **PRE-synaptic** Terminal</u>

- Auto-Receptors
  - Some axons have receptor sites for their own NT (usually inhibitory)
  - This acts as a negative feedback loop which prevents NT release if there is already a lot of the specific NT in the cleft

- Axoaxonic Synapses (Axon to Axon)
  - Presynaptic Terminal may have Receptor Sites for Inhibitory or Excitatory NT from another cell