# COGS 17 Week 3

Structure + Function of Cells in Nervous
System



# 0

### Reminders!

#### **Homework Problem Sets**

- Homework #2 is due this WED 11:59 PM!
- No late homeworks accepted

#### Midterm

- Midterm 1 is Tues, April 22 from 3-30:4:50 PM (8 days!)
- Can be taken online or in class
- Will be proctored in class

#### **Extra Credit**

- SONA
- Mnemonics
- Do all HWs → 4 extra credit points







### For Slides + Problem Sets

#### Link:

https://drive.google.com/drive/folders/1DlvXFvEKxhF3ykEaK2\_iBsNUgG0b8fS3?usp=drive\_link









# Common Features of Cells

#### Soma

- Cell body

#### Cytoplasm

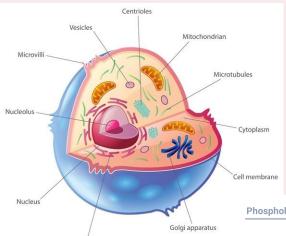
- Fluid within a cell
- Maintains structure

#### **Extracellular Fluid**

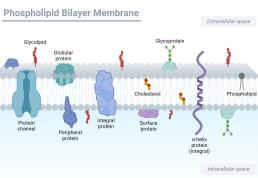
- Fluid outside of a cell

#### **Cell Membrane**

 Semi-permeable bilayer composed of lipids and proteins



Endoplasmic reticulum







# Important Organelles

#### **Nucleus**

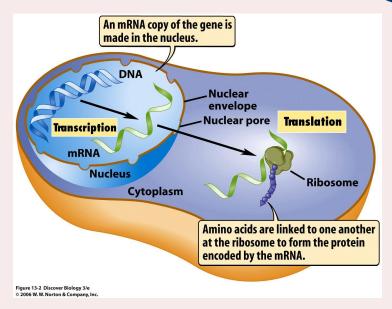
- Site for DNA storage
- "Control center"

#### Ribosomes

- Site of protein synthesis
- Receives mRNA from nucleus

#### Mitochondria

- Powerhouse of the cell!!
- Produces ATP that supplies energy for cell processes









# Cells of the Nervous System

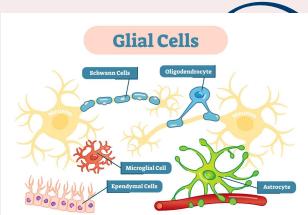
#### **Neurons**

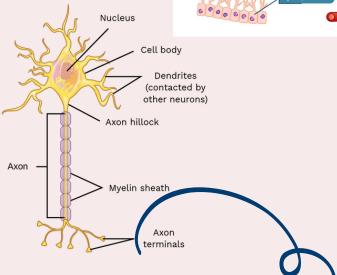
 Responsible for information transfer via modified processes and the membrane

#### Glia Cells

- Non-neural cells of the Nervous System
- Do NOT participate in info transfer
- "Glia" = "glue", holding the Nervous System together both chemically and physically
- Can regenerate unlike most neurons
- Make up 50% of the brain by weight









# Types of Glial Cells

#### **Astrocytes**

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

#### Microglia

- White blood cells of the nervous system
- Removes toxins from the brain, repairs damaged neurons

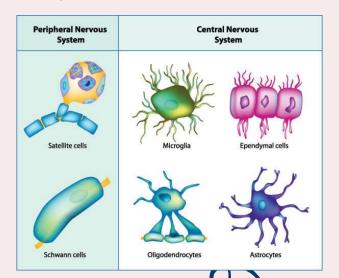
#### **Ependymal Cells**

- Lines ventricles and acts as a layer between the ventricular cavities and the parenchyma
- Secretes CSF into the ventricles





- Surrounds axons in a process called myelination in the CNS
- Schwann Cells: specialized Oligos which myelinate neurons of the PNS





### Neurons

**Specialized cells for information transfer** 

#### **Dendrites**

- Spiny protrusions from the Soma which receive incoming sign
- Site of postsynaptic membranes

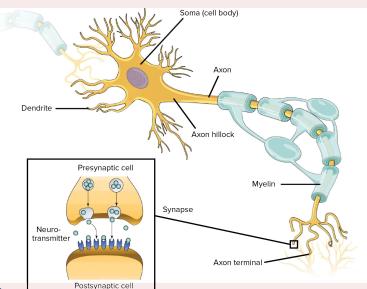
#### **Axons**

- Long fibers which reach out to other neurons
- Carries outgoing signals
- Terminates in Presynaptic Terminals (AKA terminal buttons, or end bulbs) which releases NTs into Synaptic Cleft

#### **Receptor Sites**

- Specialized areas which interact with NTs from other neurons







# The Nerve Impulse

Nature seeks a balance, or equilibrium

#### **Concentration Gradient**

 Molecules in areas of greater concentration will diffuse to areas of lesser concentration (high → low)

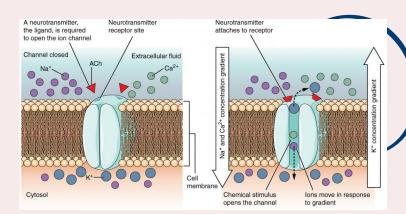
#### **Electrical Gradient**

- Like a magnet, the same charges repel whereas opposite charges attract
  - = Electrostatic Pressure

#### **Selective Permeability**

 Bilayers are typically impermeable to charged ions and large molecules (i.e. glucose)

- REMEMBER: Na+, K+, Ca++, Cl-







# Resting Potential

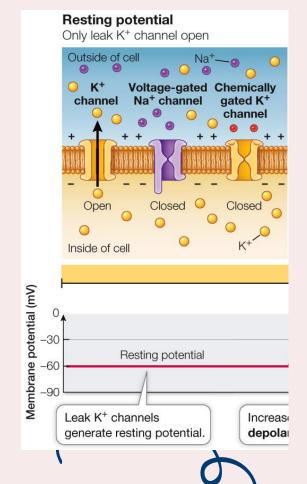
Highly polarized - ready to "fire"

#### **Membrane Potential**

 Diff in charge between the inside and outside of the cell, measured in millivolts (mV)

#### **Resting Potential**

- Most neurons have a RP of -70 mV



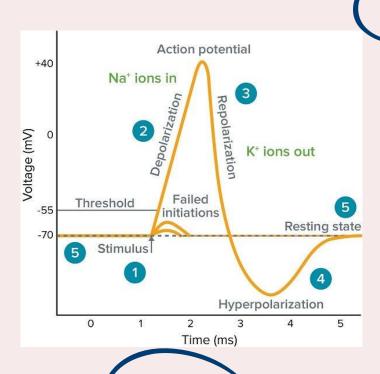




# Action Potential (AP)

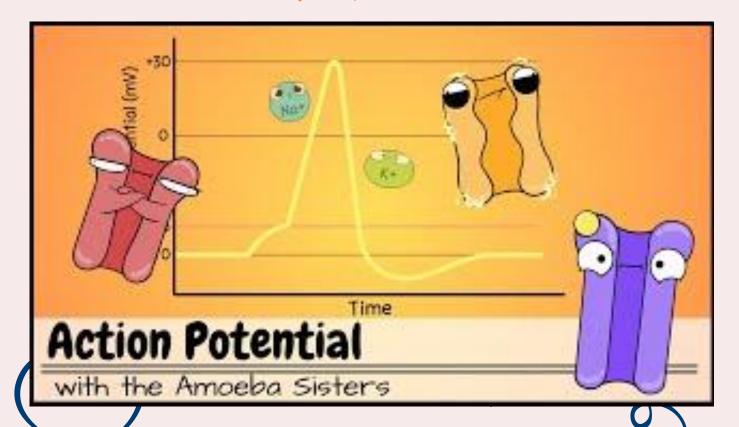
- 1. Na+ Gates at the Axon Hillock open
- 2. Na+ enters the cell, resulting in a local Depolarization (+50 mV)
  - a. More positive inside the cell
- 3. Local polarity change causes the next Na gates to open and Na+ enters cell, previous Na+ gates close
- 4. The K+ Gates at Hillock open, K+ exits cell as a result of the intracellular positive charge, resulting in a local Re-Polarization (-50 mV)
- 5. Same repeated process occurs along the axon
- 6. When "Spike" of Depolarization reaches the terminal, Ca++ enters cell & NT is released







# Action Potential (AP)





# Restoring the Resting Potential

#### **Sodium/Potassium Pump**

- Requires ATP
- Establishes resting potential by transporting 3 Na+ out and 2 K+ in

#### **Calcium Pump**

- Requires ATP
- Ejects Ca++ from Terminal

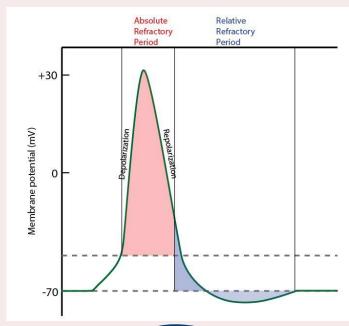
#### **Refractory Period**

- Cell cannot fire while re-polarizing

#### All-or-None Law

 AP has same amplitude and velocity, amount of NT released is fixed









# Myelination

#### **Glia Cells**

 Oligodendrocytes are wrapped around the axon, with gaps in between called the "Nodes of Ranvier"

#### **Ionic Conduction**

- Ions flow across membrane
- Slow, but stays strong

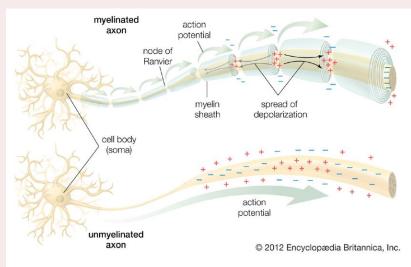
#### **Electrical Conduction**

- Electricity flows through axon under "insulation"
- VERY fast, but decays over distance

Overall, myelinated axons show Saltatory ("Jumping")
Conduction





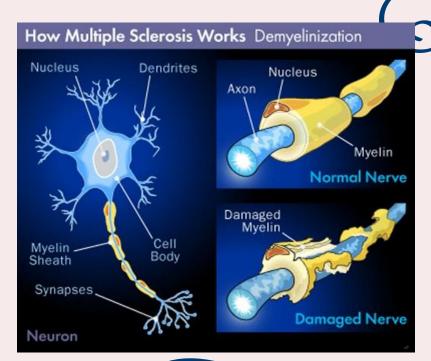






**Characterized by damaged myelin that degenerates** gradually

**Damaged nerves cannot carry messages** 









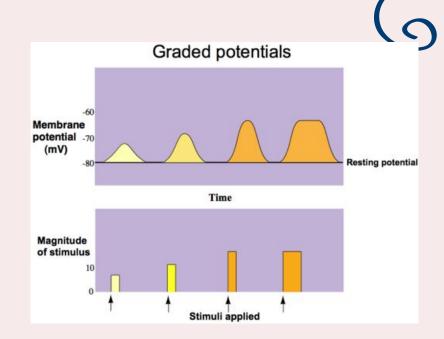
## **Graded Potentials**

**Not all neurons show APs** 

Cells that fire "Graded Potentials" may release MORE or LESS NTs

#### Ex. Hair Cells that contain auditory receptors

- Soft sound, cilia move a little  $\rightarrow$  a little NT is released
- Loud sound, cilia move a lot  $\rightarrow$  a lot of NTs are released









# The Synapse

Presynaptic cell + Synaptic Cleft + Postsynaptic cell = The Synapse

Presynaptic cells release NTs into the cleft via Exocytosis

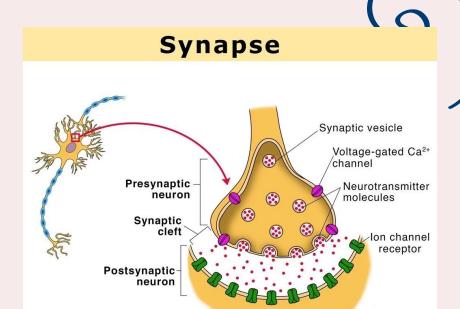
- NTs are packaged into vesicles

Influx of Ca initiates 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









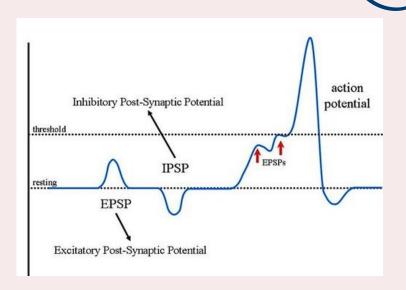
# Postsynaptic Polarity

#### **EPSP**

- Cell becomes HYPO-polarized (more positive)
- Membrane potential also rises, increasing likelihood of initiating an AP

#### **IPSP**

- Cell becomes HYPER-polarized (less positive)
- Membrane potential decreases, further away from threshold makes it less likely to fire an AP









### **Summation**

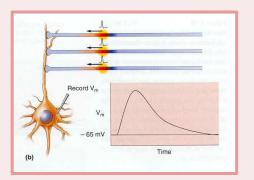
Multiple excitatory and inhibitory inputs converge on each cell

#### **Temporal**

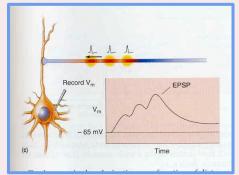
- When one (or more) cells repeatedly stimulate another in rapid succession

#### **Spatial**

- When multiple cells converge on a single cell at the same time













# Postsynaptic Mechanisms

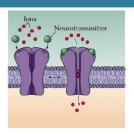
#### **Ionotropic**

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

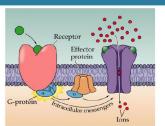
#### Metabotropic

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

#### Ionotropic versus Metabotropic Receptors



- Fast on/off
- All or none on/off
- Triggers action potentials
- Made up of multiple interchangeable subunits



- Slower off /off
- Can amplify or dampen signals
- Triggers multiple post-synaptic events, including action potentials
- Monomers







Neurotransmitter Examples w/ Functions

Neurotransmitter	Functions
Acetycholine (Ach)	All neuro-muscular junctions     Cortical arousal
GABA	Most common inhibitory NT     Regulate anxiety
Glutamate	<ul><li>Most common excitatory NT</li><li>Learning</li><li>Perception</li><li>Schizophrenia</li></ul>
Serotonin (5HT)	<ul><li>Often acts as a neuromodulator</li><li>Mood regulation, sleep, perception</li></ul>
Dopamine	<ul><li>Reinforcement</li><li>Attention</li><li>Motor control</li></ul>
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





# Agonists vs. Antagonists

**Agonists** → **Increases** effect of a NT

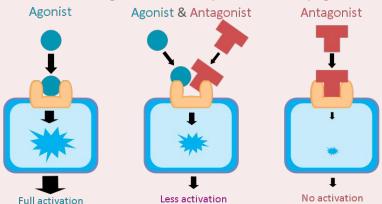
- Ex. Acetylcholinesterase
  - Enzyme which breaks down ACh in the cleft
- Ex. Black Widow Spider venom causes massive release of NT (ACh)
- Ex. Serotonin Reuptake
  - Prosac (antidepressant): serotonin reuptake inhibitor (SSRI), increasing NT's duration in the cleft

**Antagonists** → **Decreases or inhibits** effect of a NT

 Ex. Reserpine prevents NTs from being packaged into vesicles



Antagonists - Drugs that occupy receptors but do not activate them Antagonists block receptor activation by agonists.









- 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
  - a. Ex. LSD binds to Serotonin sites
- 4. Some NTs may require Hours/Days to replenish
  - a. 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 (Ex. L-DOPA)

#### Exceptions: Receptor Sites on PRE-synaptic Terminal

- 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)



