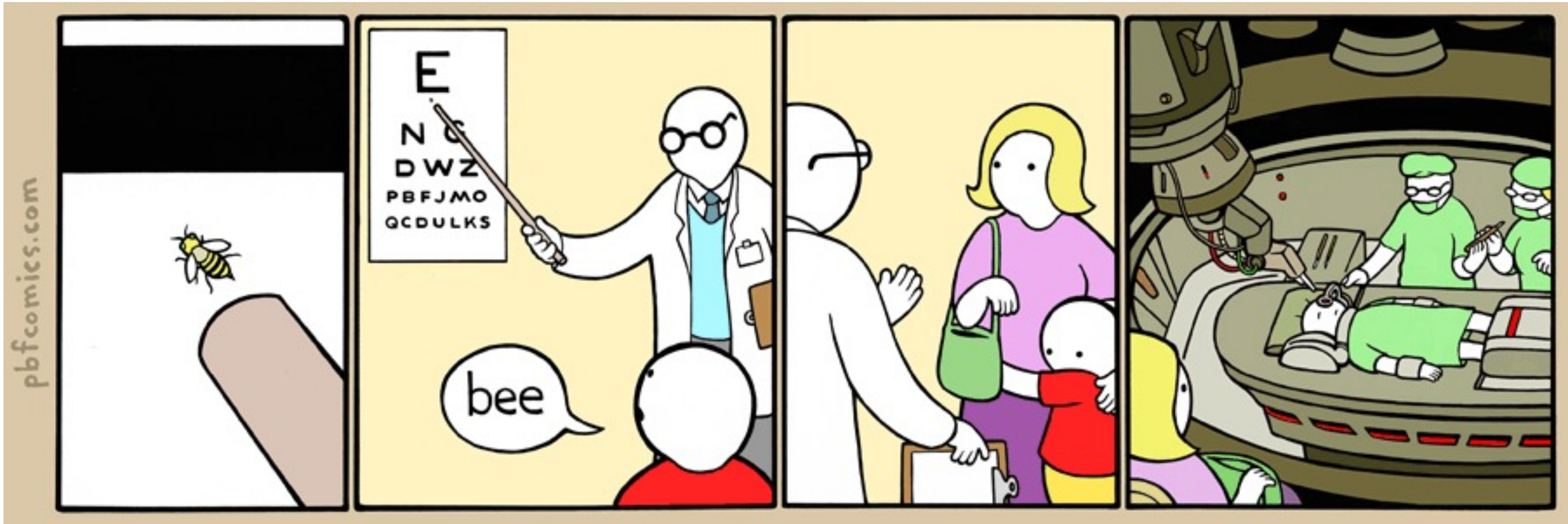
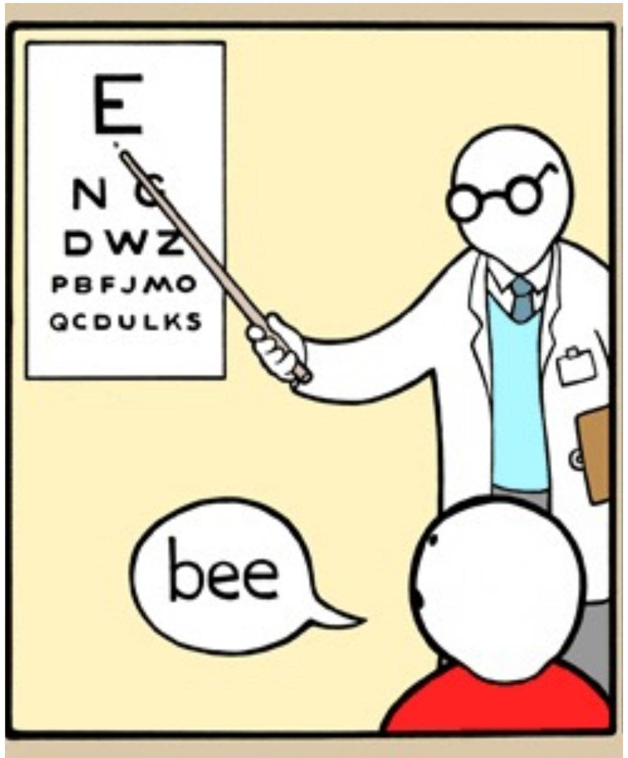
# PSYC301: Neural communication

Jay Hosking, PhD

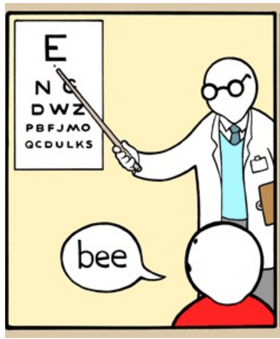


# Overview

I. Within one cell
   a. The resting membrane potential
   b. Post-synaptic potentials
   c. Action potentials
   d. Action potential conduction
II. Between cells
   a. The synapse and neurotransmission
   b. Receptors

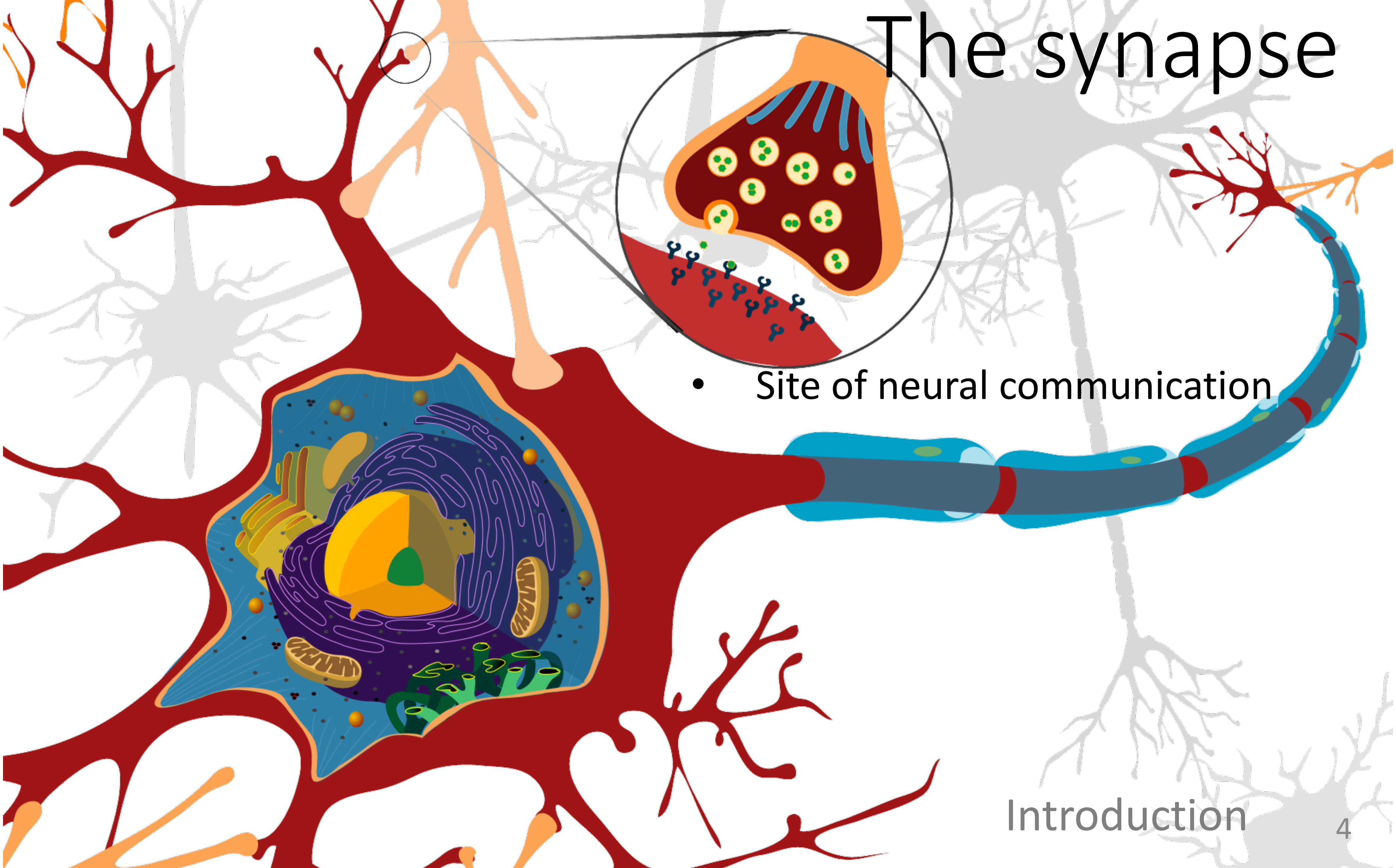


# Learning objectives



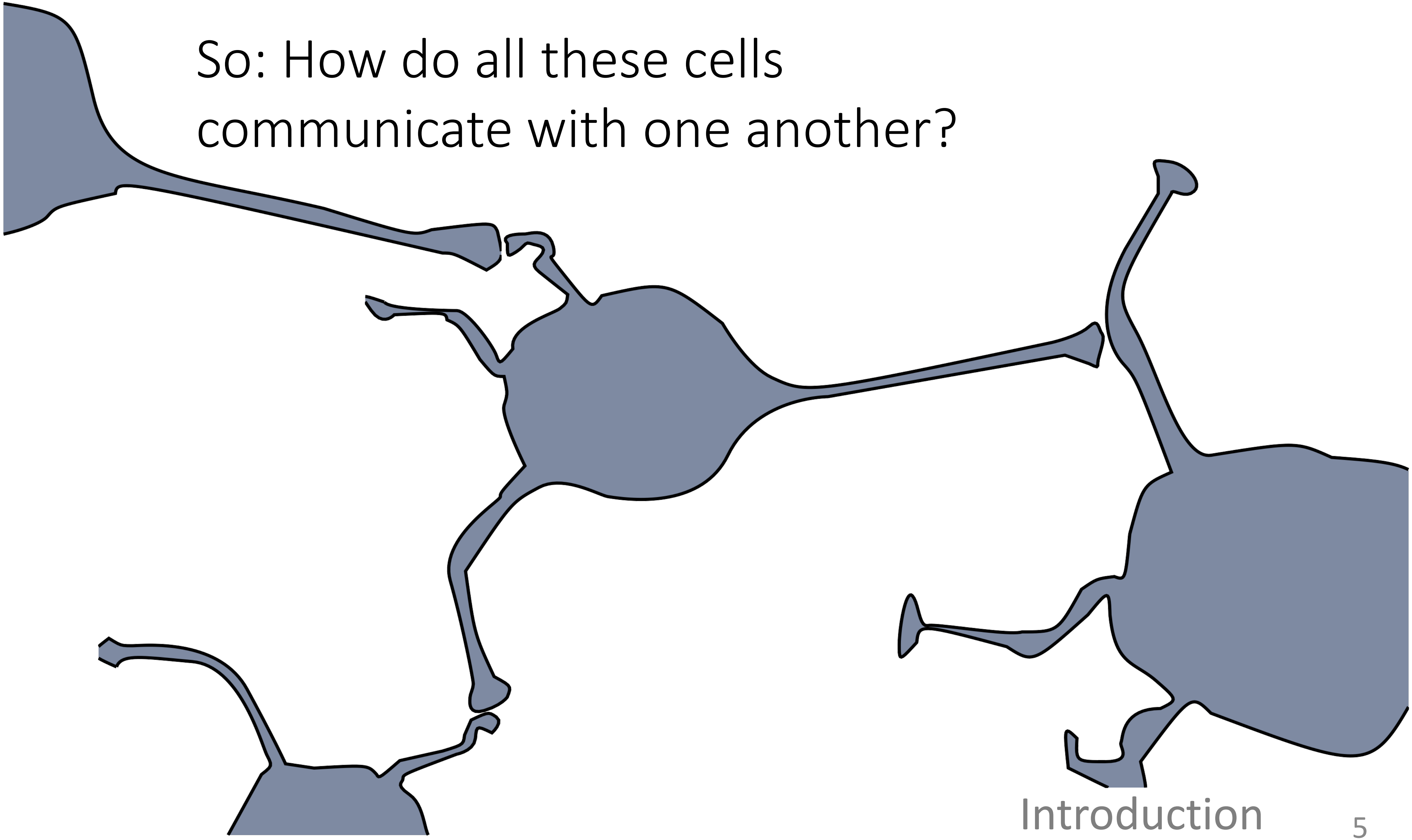

1. How is neural communication measured and recorded?
2. Identify the ions that are important for neural communication, including their electrical properties.
3. What is the resting membrane potential and why is it important? What happens if the resting membrane potential is disrupted?
4. Describe the proteins involved in maintaining the resting membrane potential.
5. What are post-synaptic potentials? Describe the two main forms they take, their effects, and what causes them.
6. What is an action potential? Where does it occur? What proteins cause it?
7. Describe conduction of the action potential, and strategies to increase speed.
8. Describe the axon terminal, and the effect of an action potential on the bouton.
9. What are receptors? Be sure you can explain effects, types, and location.
10. How is a signal between cells ended?
11. (Optional.) Read chapter 4 of Pinel & Barnes for some extra help on the material.

# The synapse

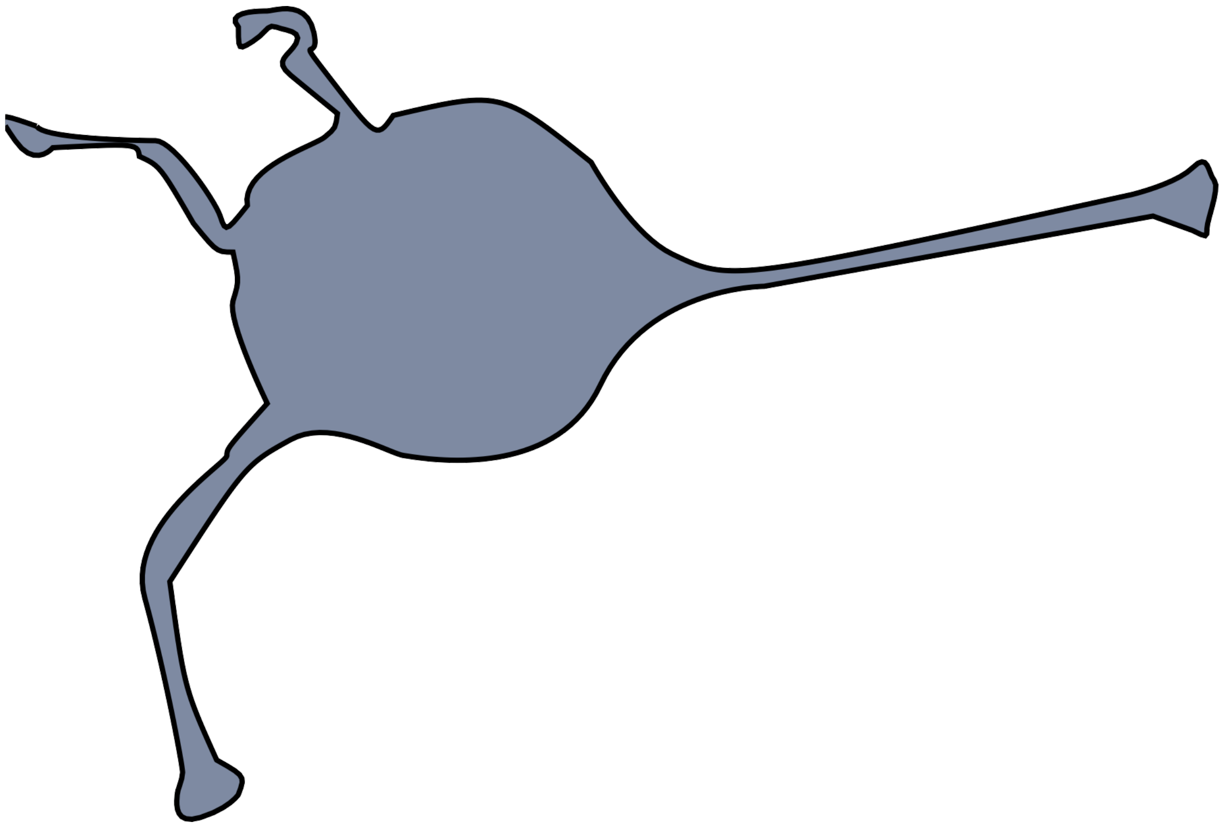


- Site of neural communication

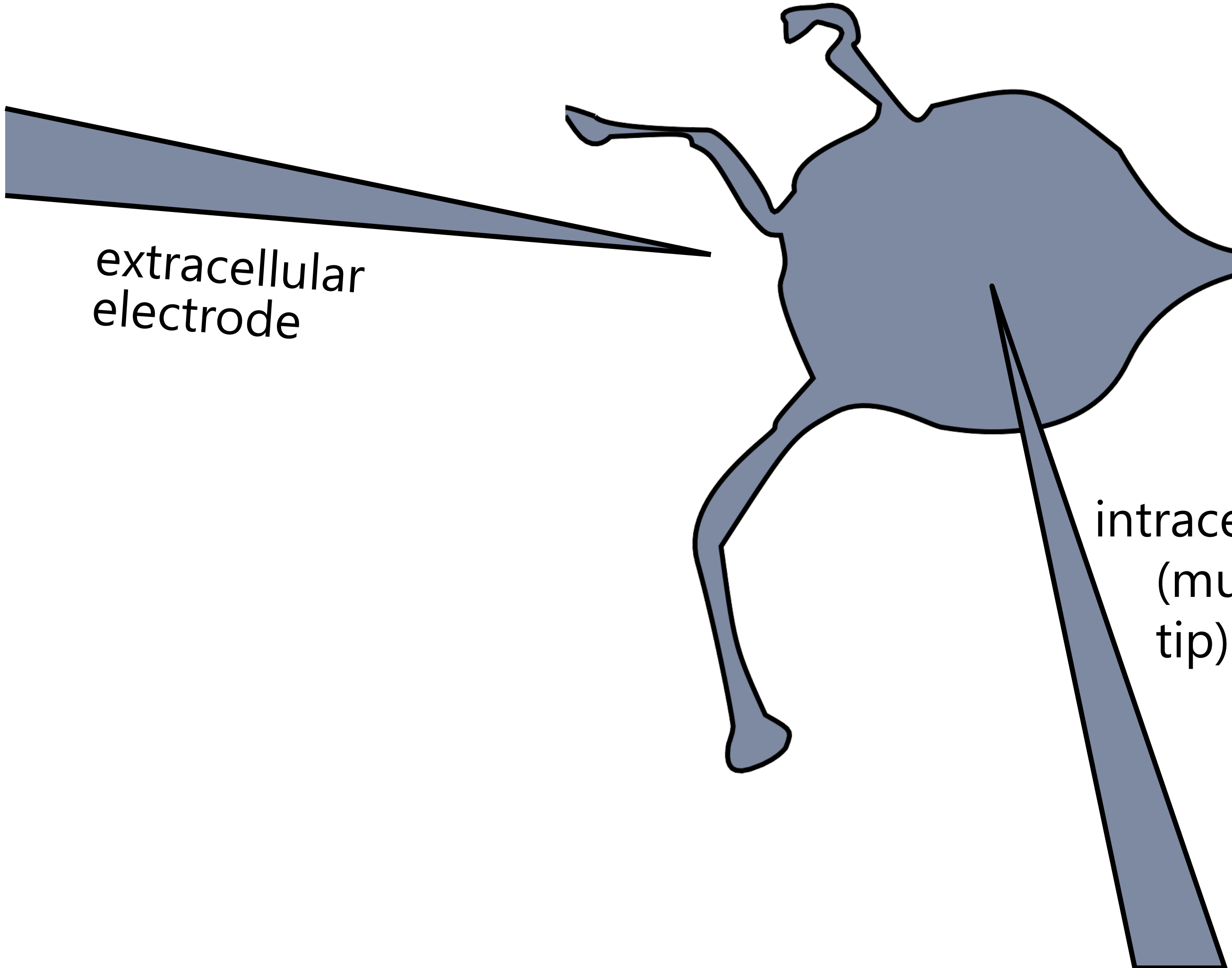
# So: How do all these cells communicate with one another?

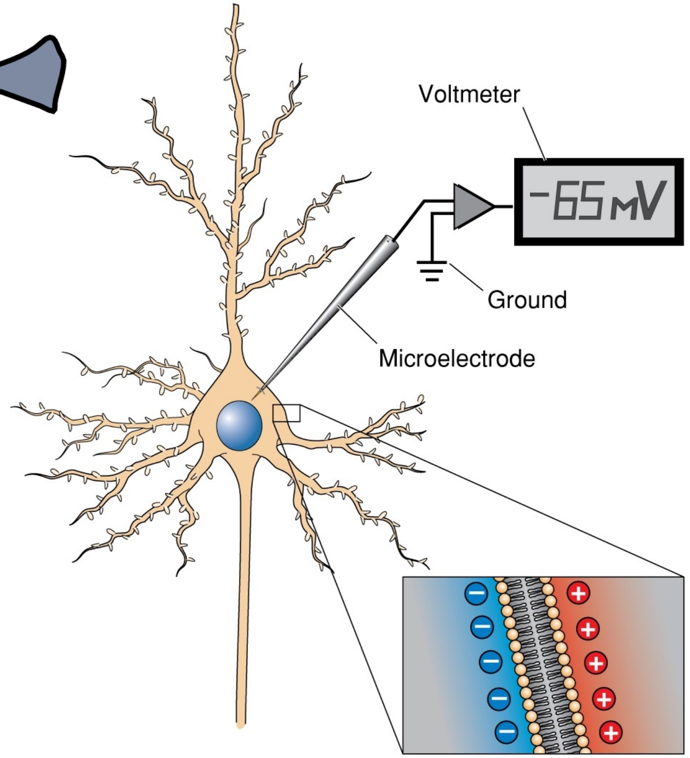


# As a first step: How does communication occur within a single neuron?



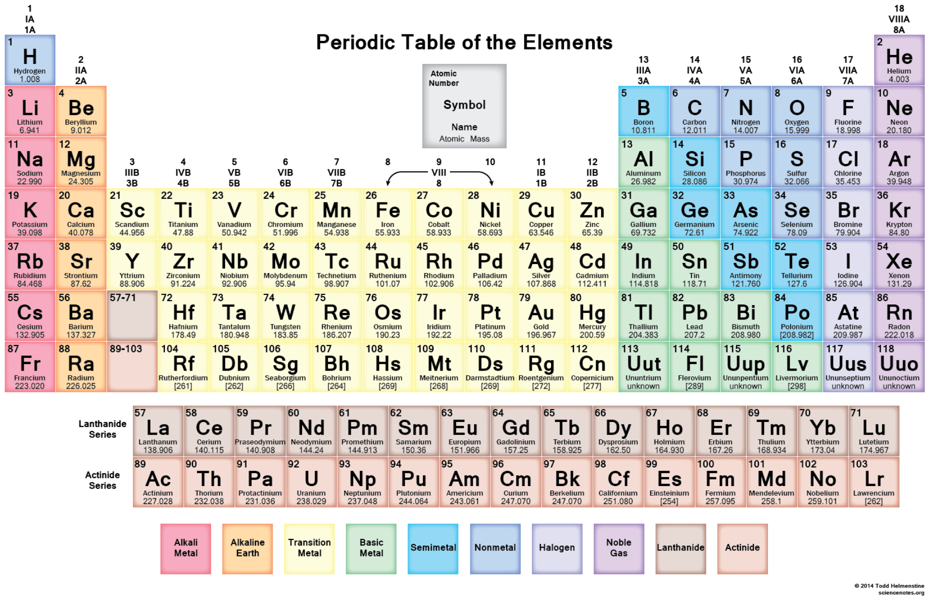
# A healthy neuron has a resting membrane potential (voltage) of between -60 and -80 mV; you'll hear me say -65mV or -70mV often.

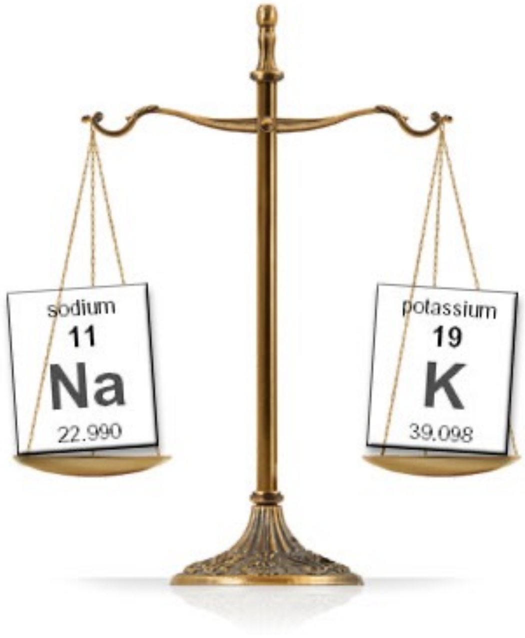


# Neuronal communication is chemical

1. Primarily the result of two ions, sodium (Na+) and potassium (K+)
2. Ions move into or out of the cell, but not freely



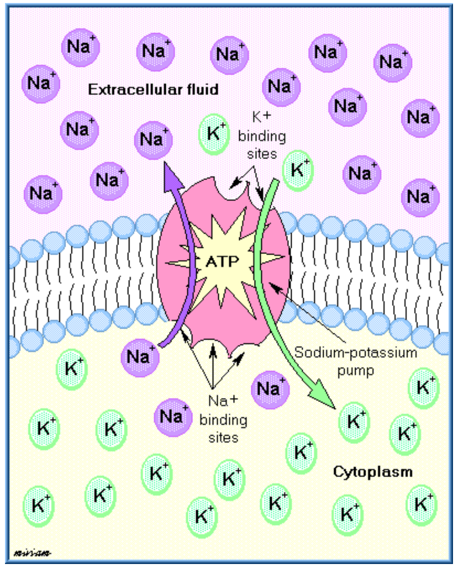

Resting membrane potential

# Neuronal communication is electrical

1. Ions are positively and negatively charged (Na+ and K+ are both positive, as per "+")
2. As they move into or out of cell, they change the *potential* (voltage) at the membrane

   Note: absence of pos. is neg.!

   i.e. remove a pos., leave a neg.



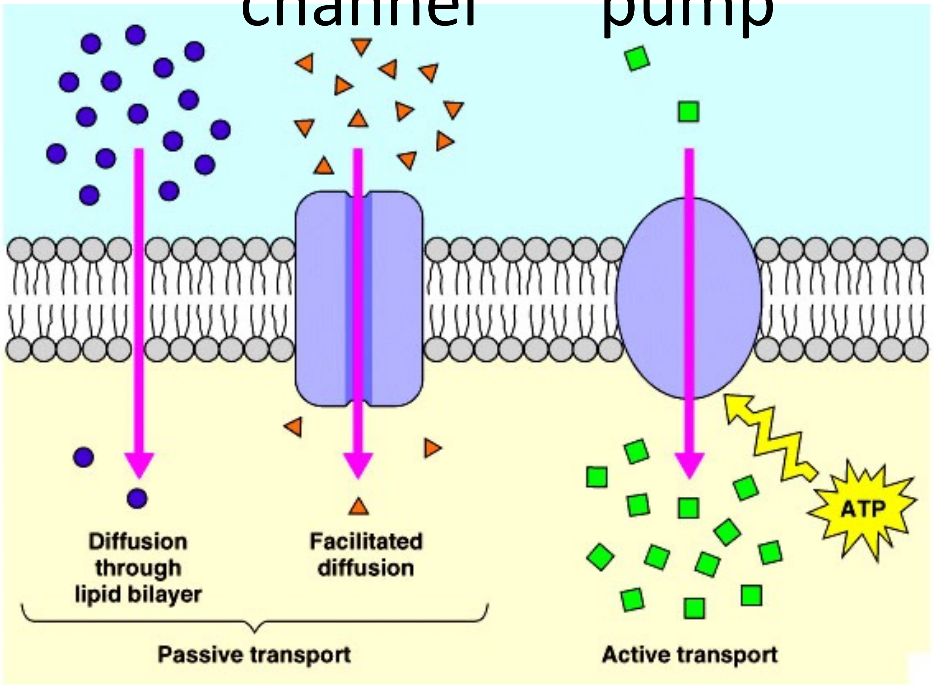

And yes, neurotransmission is also chemical, but we'll talk about that one later!

Resting membrane potential

# Channels and pumps

- Remember, the plasma membrane is an effective barrier!
- Only certain molecules/ions are permitted to cross membrane, via *channels* and *pumps*
  - Channels: allow *passive* diffusion (i.e. along chemical gradient)
  - Pumps: actively push ions against their chemical gradient
    - Requires energy (ATP)

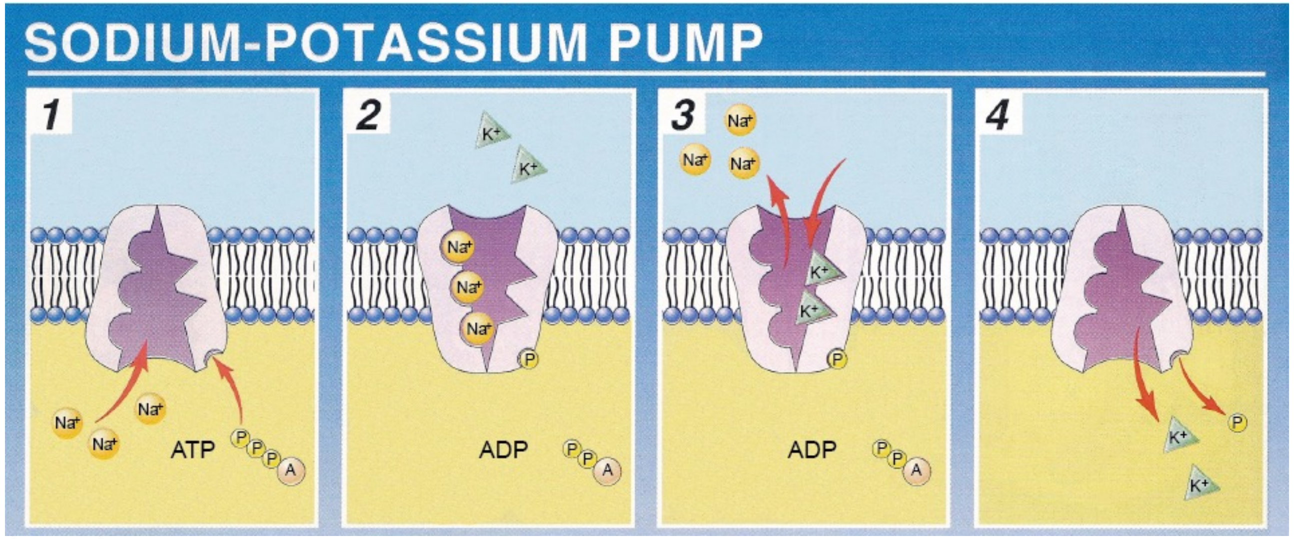
channel pump

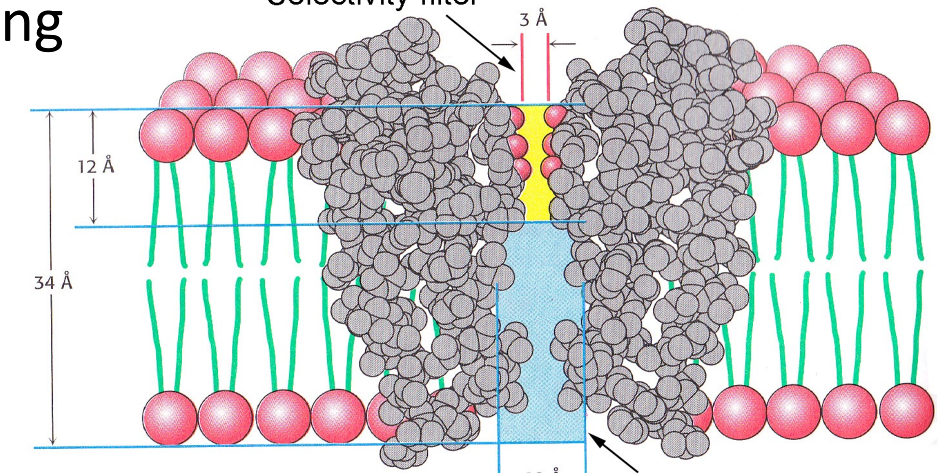


# Proteins of the resting membrane potential

1. **Sodium-potassium pumps**
   - Use 2/3rds of all brain energy (ATP)
   - 3 Na+ out, 2 K+ in
2. **Potassium “leak” channels**
   - Require no energy

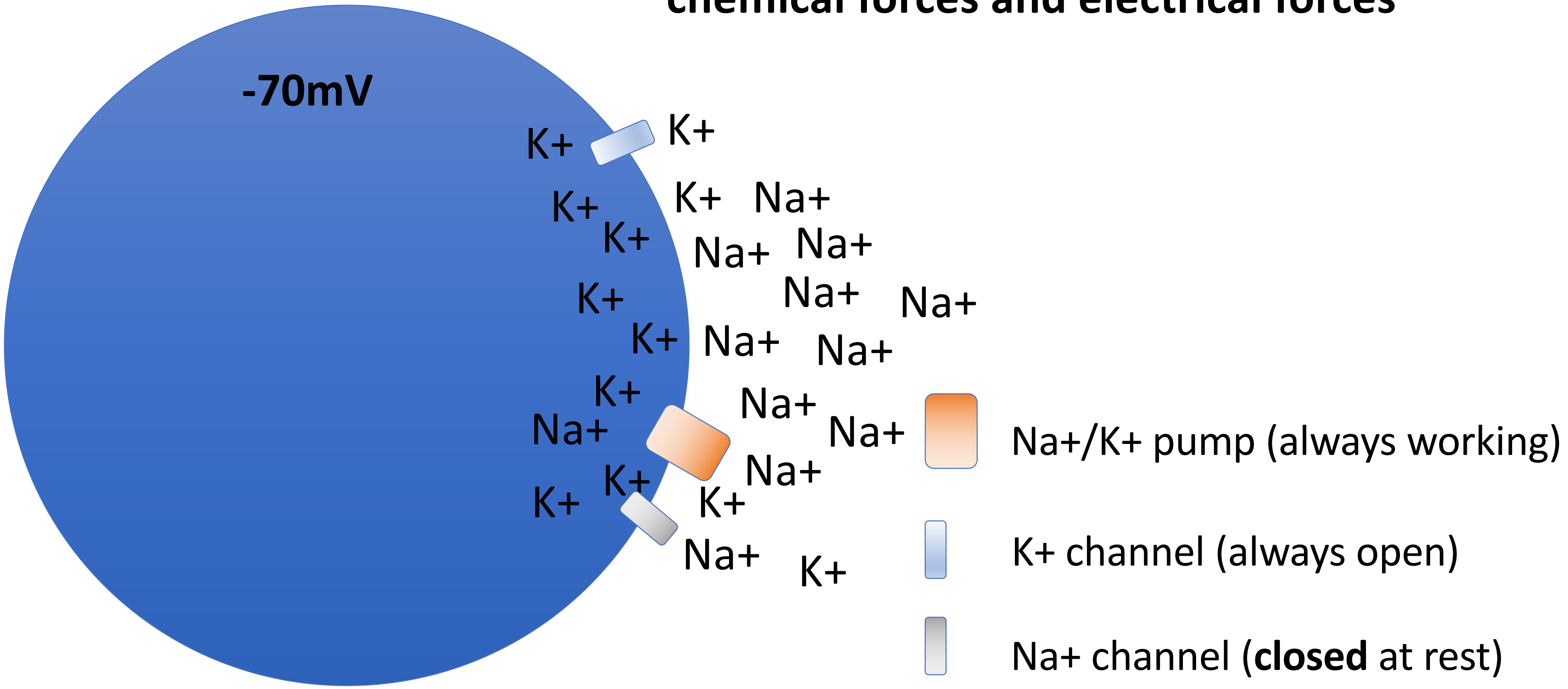
- Both are embedded in membrane
- Both are constantly working



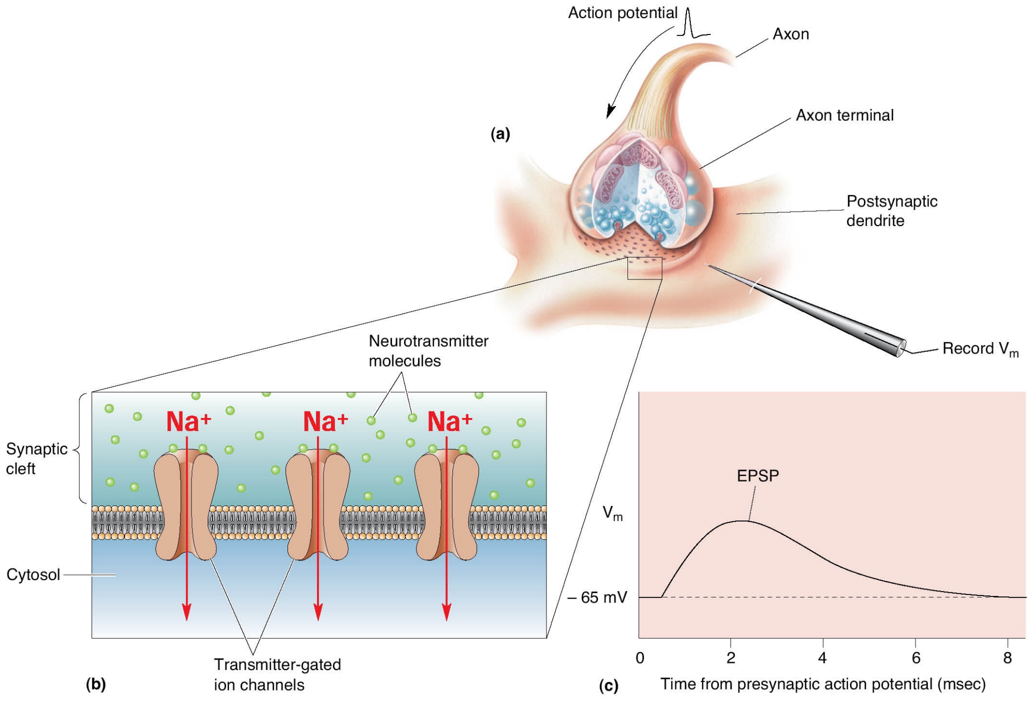
# Cells are *polarized*

**The RMP is an equilibrium between chemical forces and electrical forces**



When a neurotransmitter binds to a receptor, it can have one of two localized effects:

**1. Depolarize the membrane** (e.g. from -70 to -67mV) → excitatory post-synaptic potential (EPSP) → increase likelihood of an action potential (AP)
**2. Hyperpolarize the membrane** (e.g. from -70 to -72mV) → inhibitory post-synaptic potential (IPSP) → decrease likelihood of AP




*Why* transmitters/receptors cause these effects will be talked about once we reach the synapse! (But the image to the left is a hint.)

## Postsynaptic potentials

When a neurotransmitter binds to a receptor, it can have one of two localized effects:

**1. Depolarize the membrane** (e.g. from -70 to -67mV) → excitatory post-synaptic potential (EPSP) → increase likelihood of an action potential (AP)
**2. Hyperpolarize the membrane** (e.g. from -70 to -72mV) → inhibitory post-synaptic potential (IPSP) → decrease likelihood of AP

The transmission of postsynaptic potentials (PSPs) is **graded, rapid, and decremental**: PSPs travel like an electrical signal along an uninsulated wire.

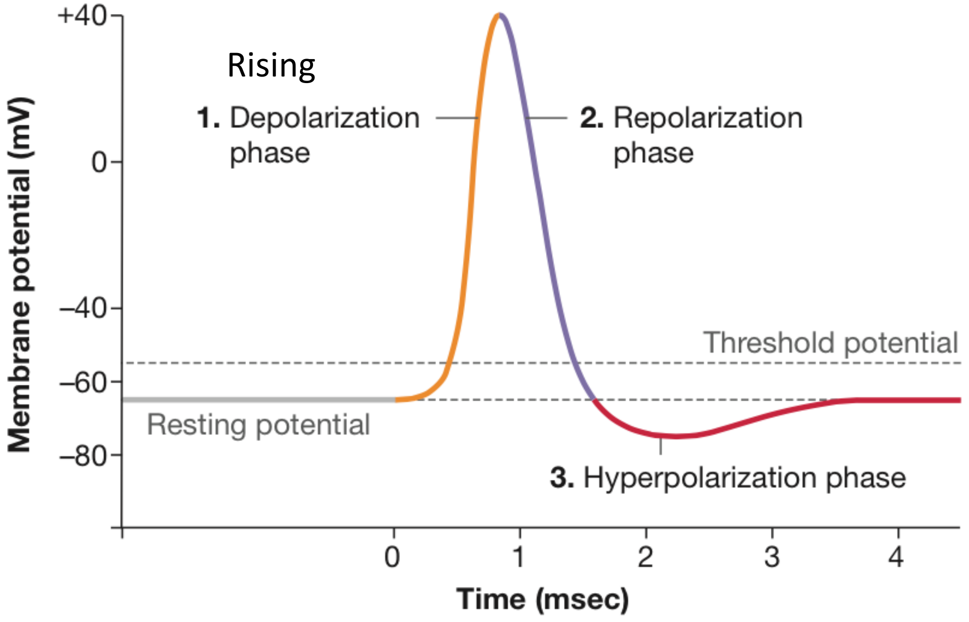
# AP Generation

If the sum of the EPSPs and IPSPs that reaches the **axon initial segment** is sufficient to depolarize the membrane there above its **threshold of excitation,** then an **action potential** (AP) is generated

The AP is a massive, brief reversal of the membrane potential (e.g. from -70 to +55 mV)

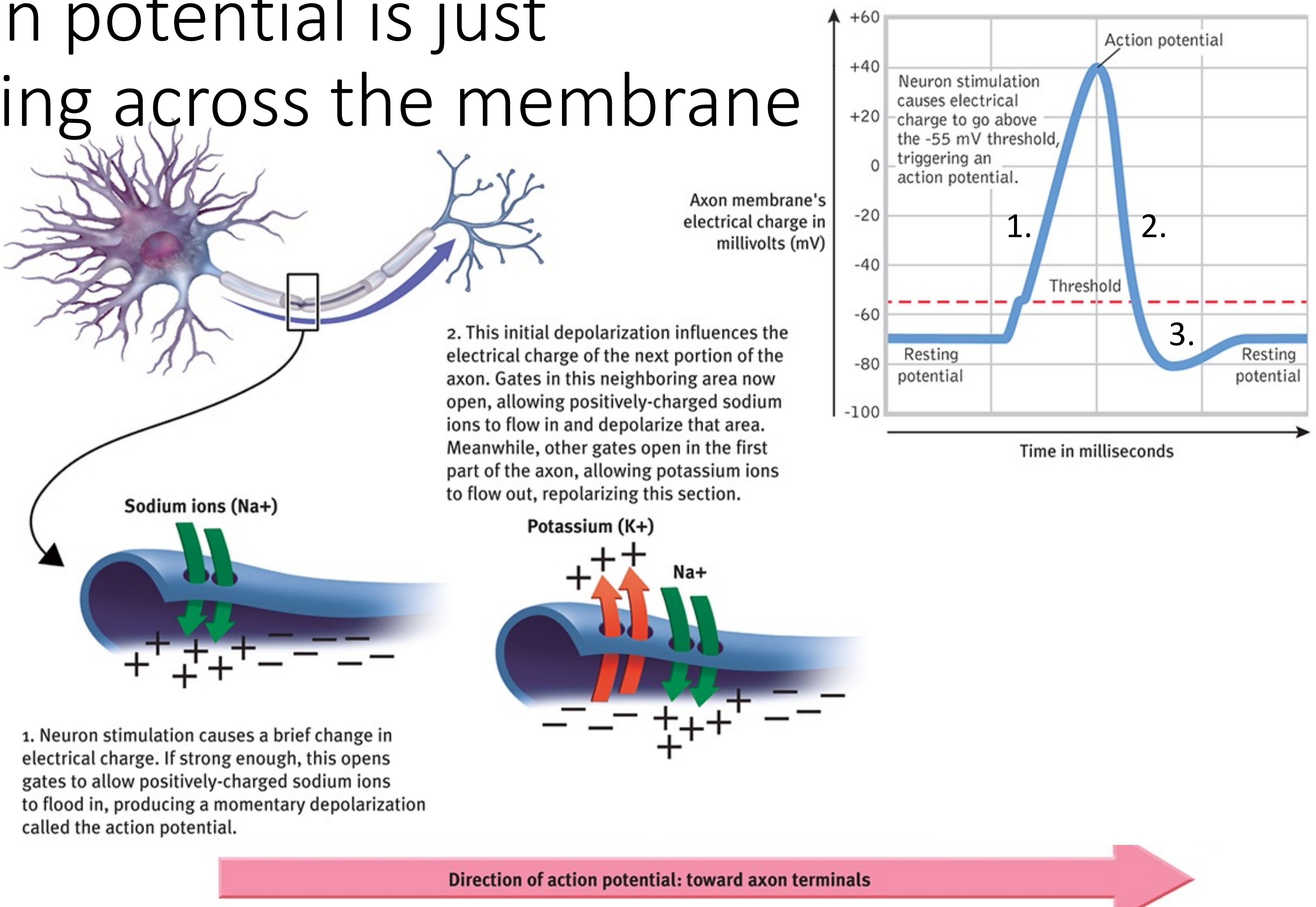
The AP is an all-or-none phenomenon
i.e. Not graded
Always same size/shape

The threshold of excitation is simply where **voltage-gated** Na+ channels open!




Action potentials

# The action potential is just ions moving across the membrane




Action potentials

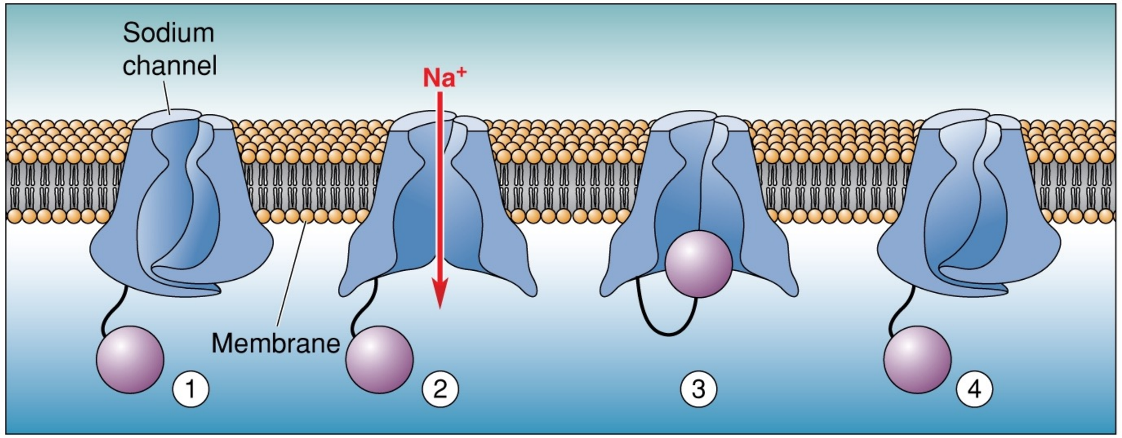


**FIGURE 2.4** Myers/DeWall, *Psychology*, 12e, © 2018 Worth Publishers

# Proteins of the action potential

1. **Voltage-gated sodium channels ($Na_v$)**
   - Open at the threshold of excitation
   - Have an "auto-shutoff" after about 1ms
   - Responsible for rising phase of AP
2. **Potassium channels (two types)**
   - Leak channels, same as before, always open
   - Also voltage-gated channels ($K_v$) that start to open during the rising phase of the AP
   - Responsible for repolarization & hyperpolarization

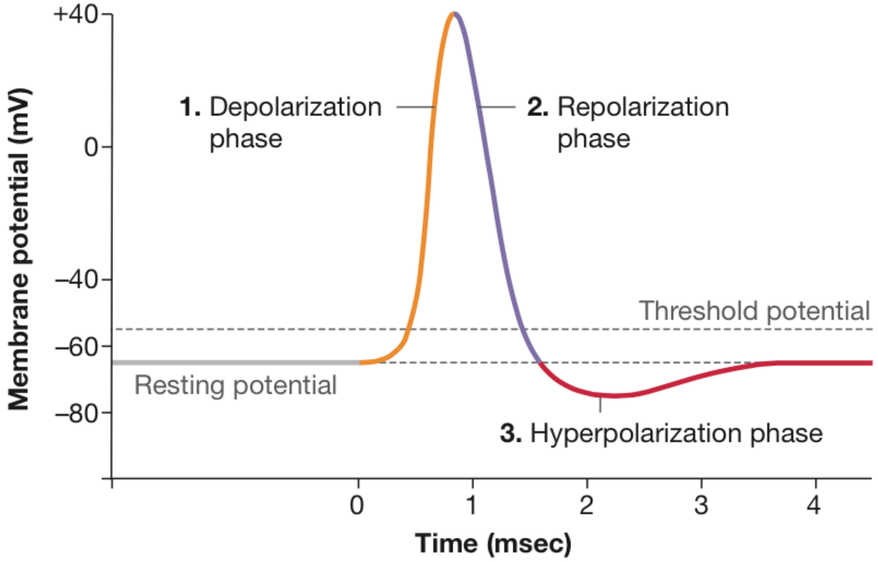
- Note that the sodium-potassium pump is too slow to play a role in the AP!

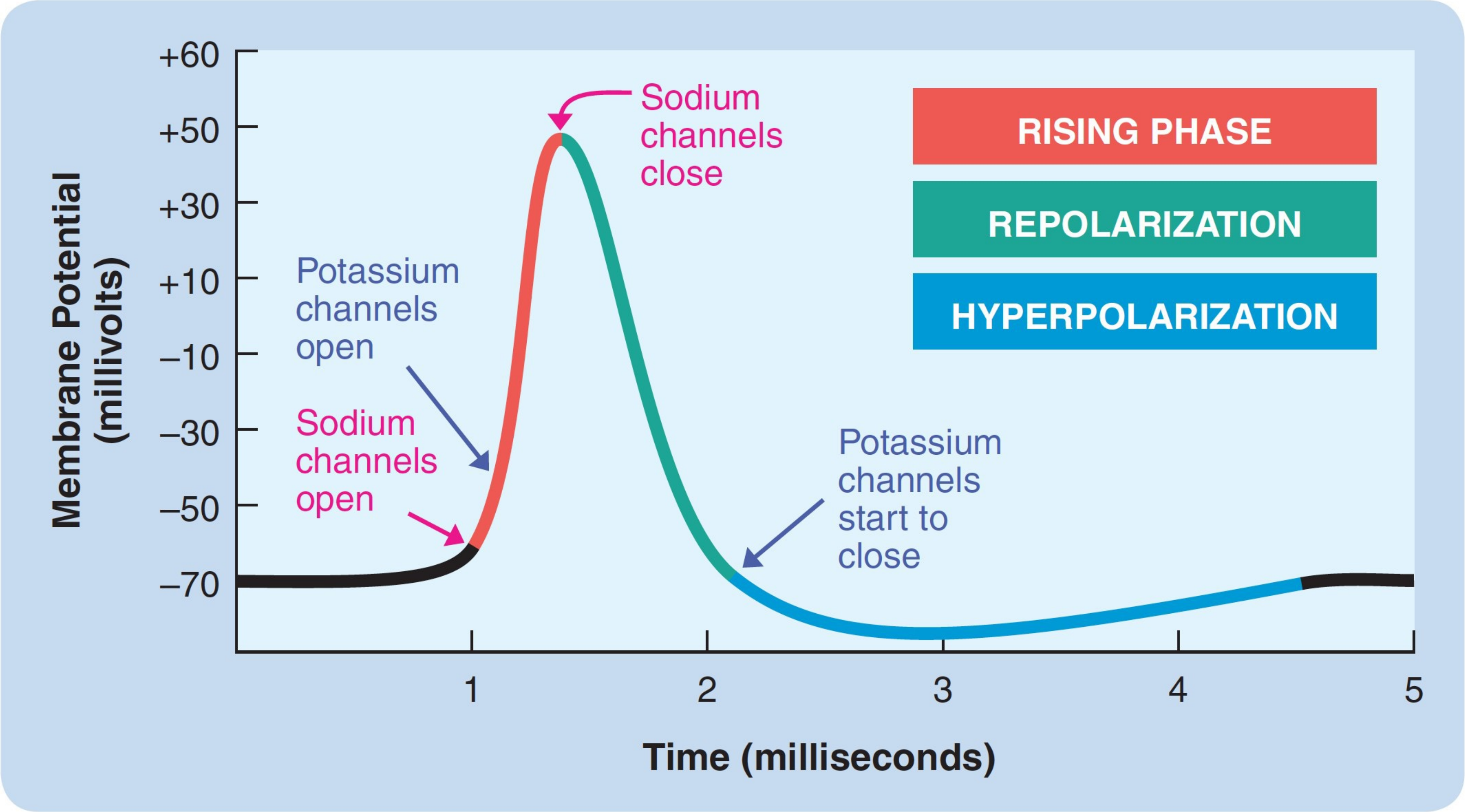



(c)



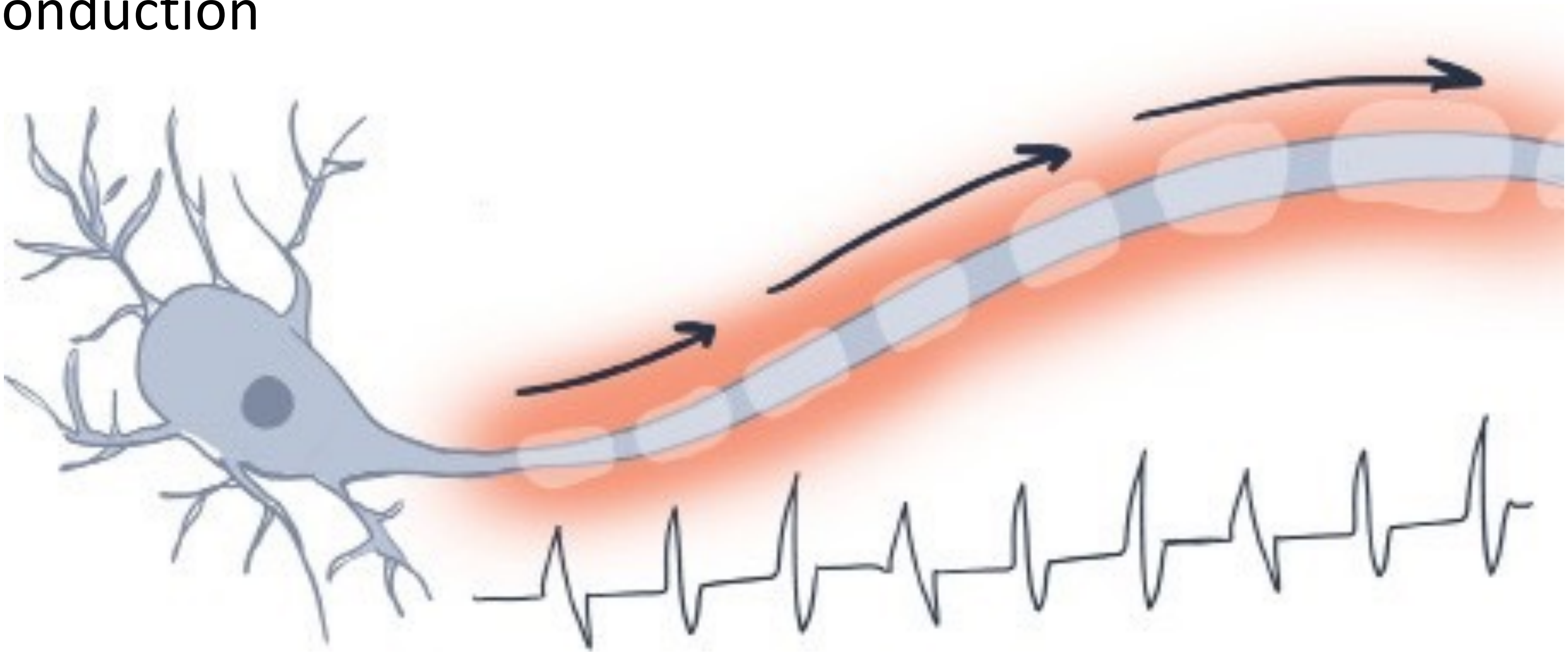
$Na_v$





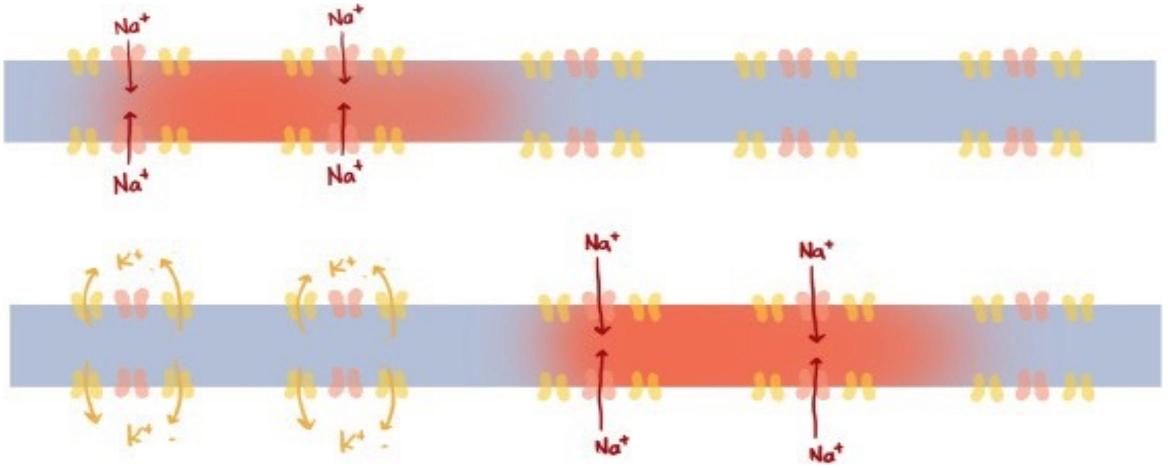

Action potentials

The AP starts at the initial segment and travels to the terminal: conduction

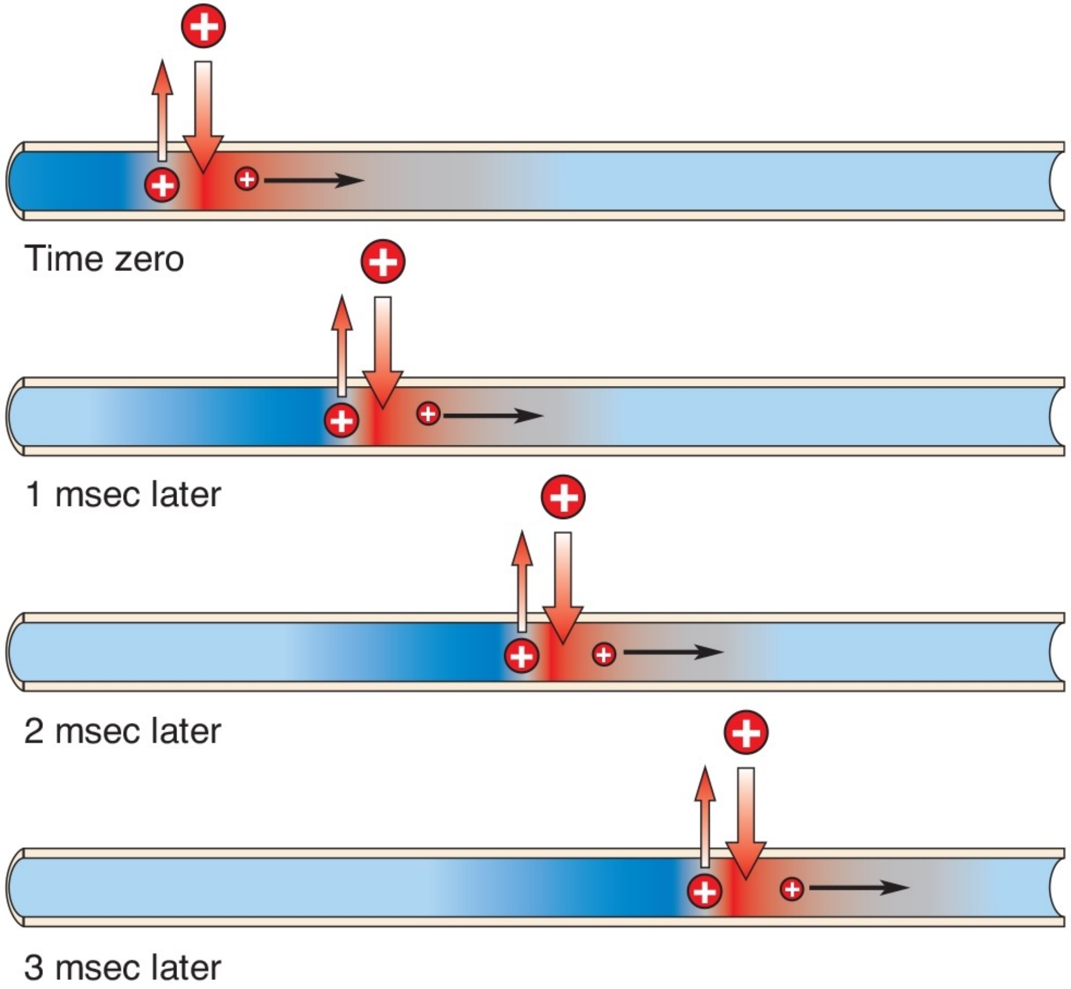


AP looks the same all down axon
i.e. it does not decay (it is constantly regenerated)

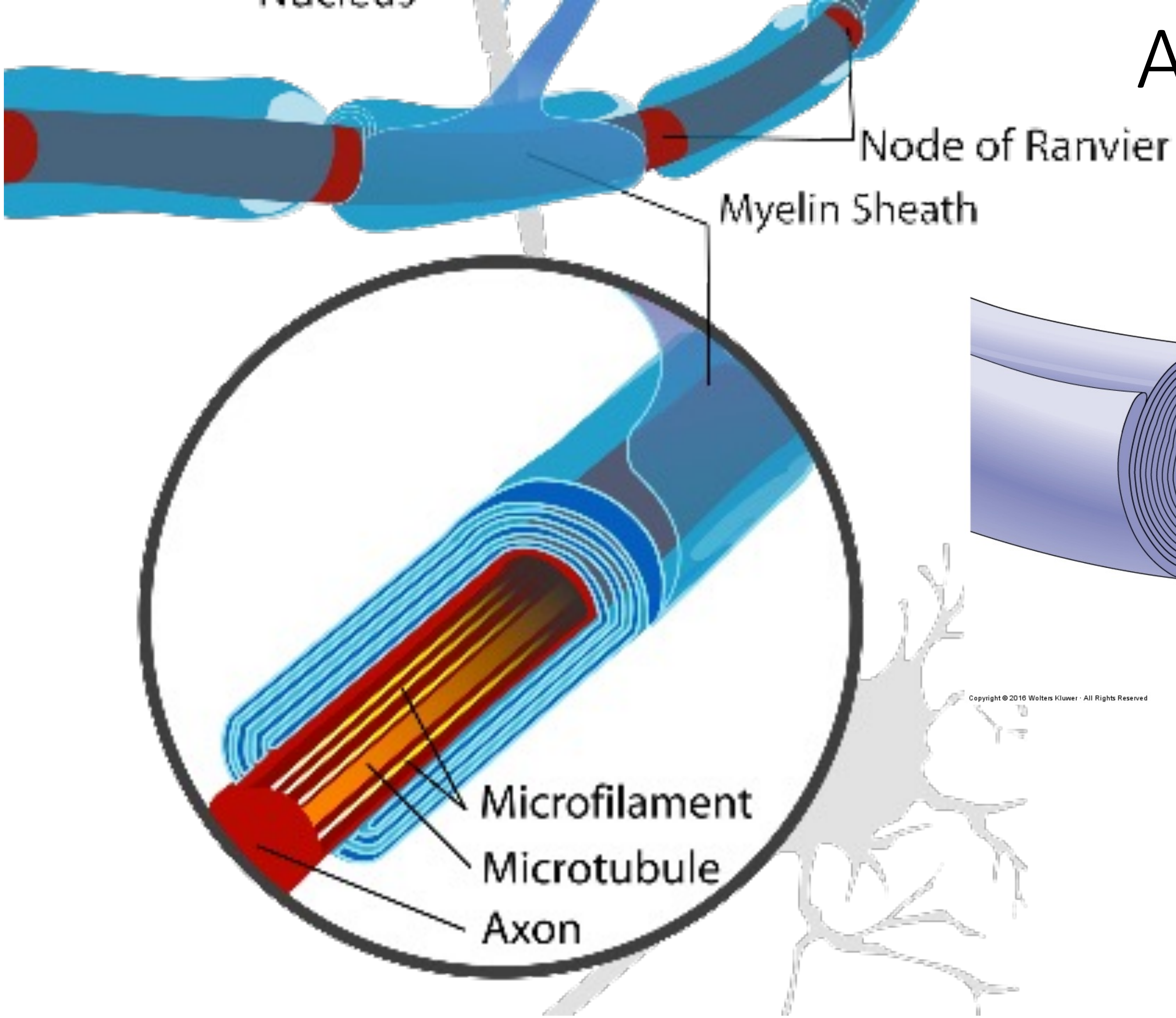
# Conduction in an Unmyelinated Axon

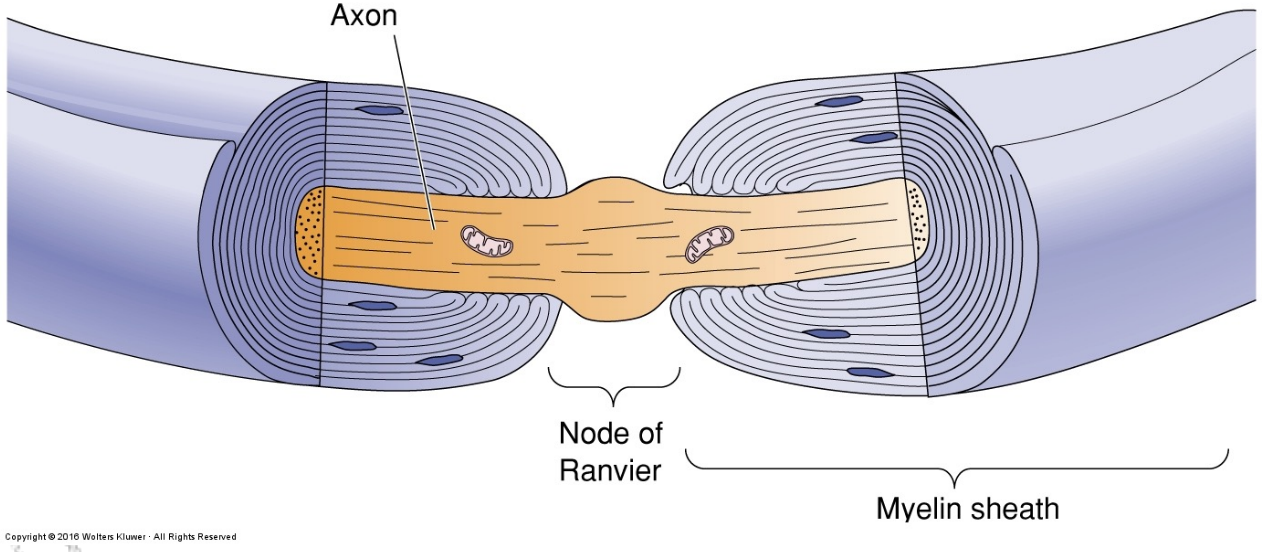



- Na+ channels ($Na_v$) are present along axon
- Unmyelinated axons: $Na_v$ everywhere
- Conduction speed is limited by number of $Na_v$ (like doors in a hallway)
- But if you have too few $Na_v$, the AP decays



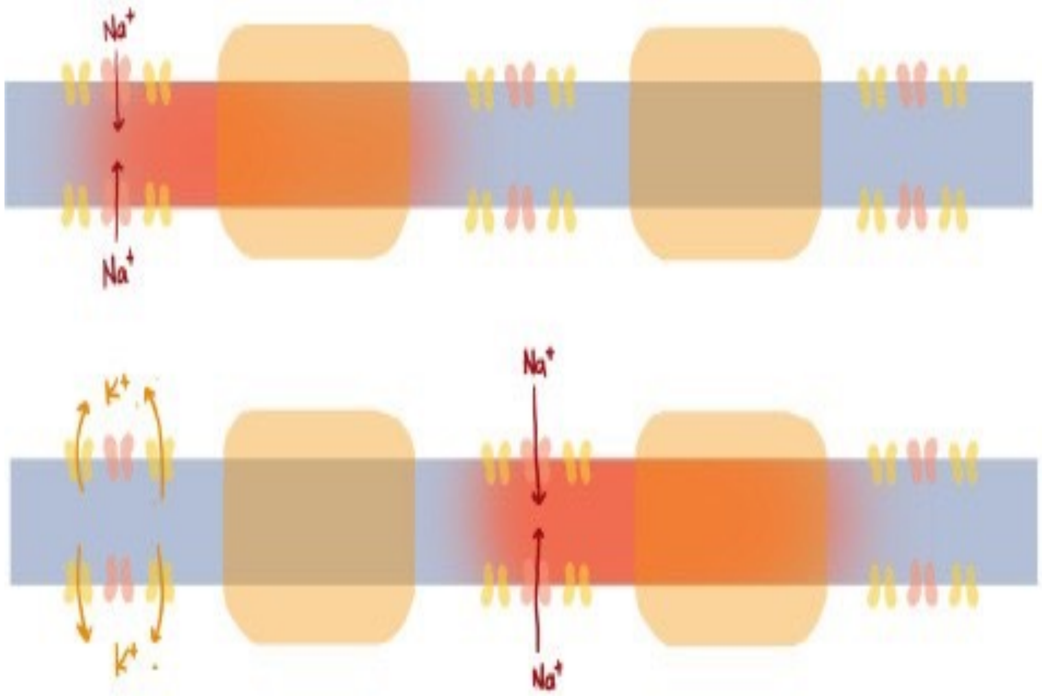
# Axon Myelination

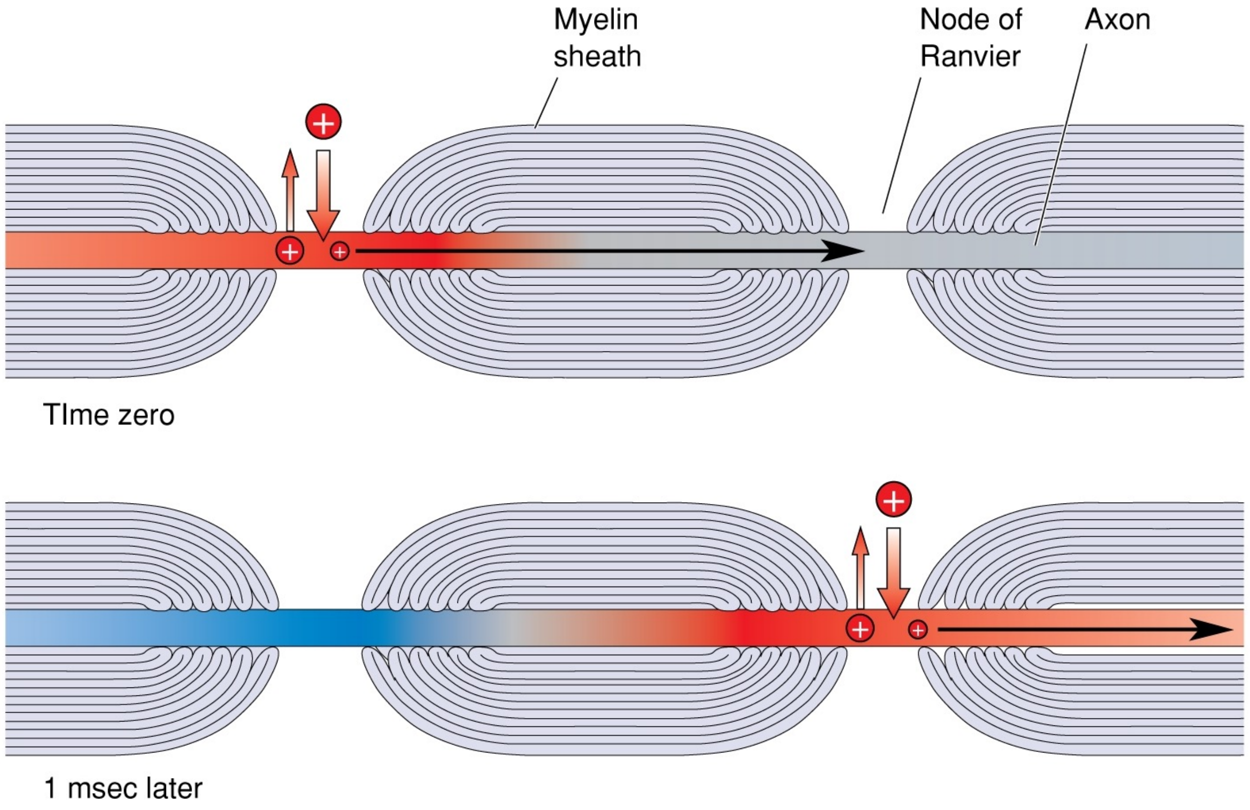



Myelin causes the AP to decay less quickly as it spreads

# Conduction in a Myelinated Axon



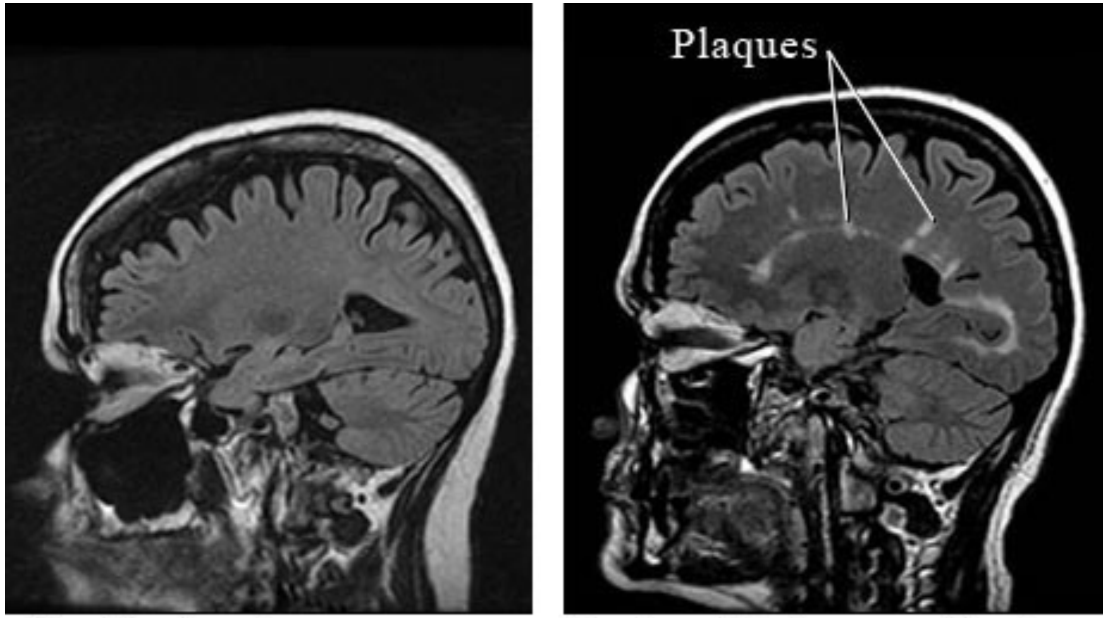






- $Na_v$ are present all along the axon
- Unmyelinated axons: $Na_v$ everywhere
- Myelinated axons: $Na_v$ only at the Nodes of Ranvier
  - Fewer ”doors”, faster conduction down the “hall”

# Related to myelin: Multiple Sclerosis (MS)

- A disorder that progressively damages myelin
- 55-75K in Canada (3 new/day)
- Canadians have one of the highest rates of multiple sclerosis in the world
- Likely some connection to Epstein-Barr virus (a type of herpes virus)
- More on this later



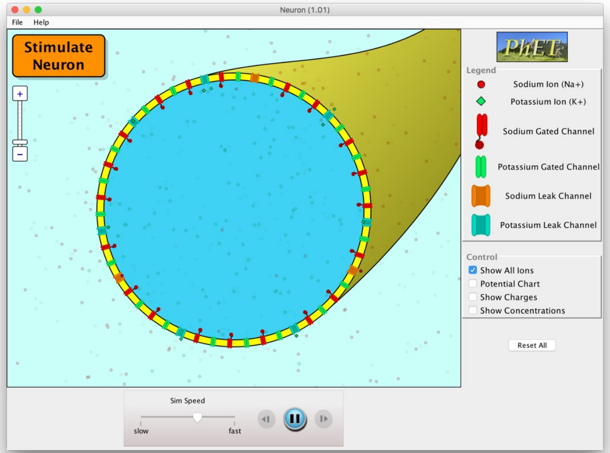

Healthy brain

Brain with damage (lesions or plaques) caused by MS

Image from:
myhealth.alberta.ca/health/Pages/conditions.aspx?hwid=zm6056&

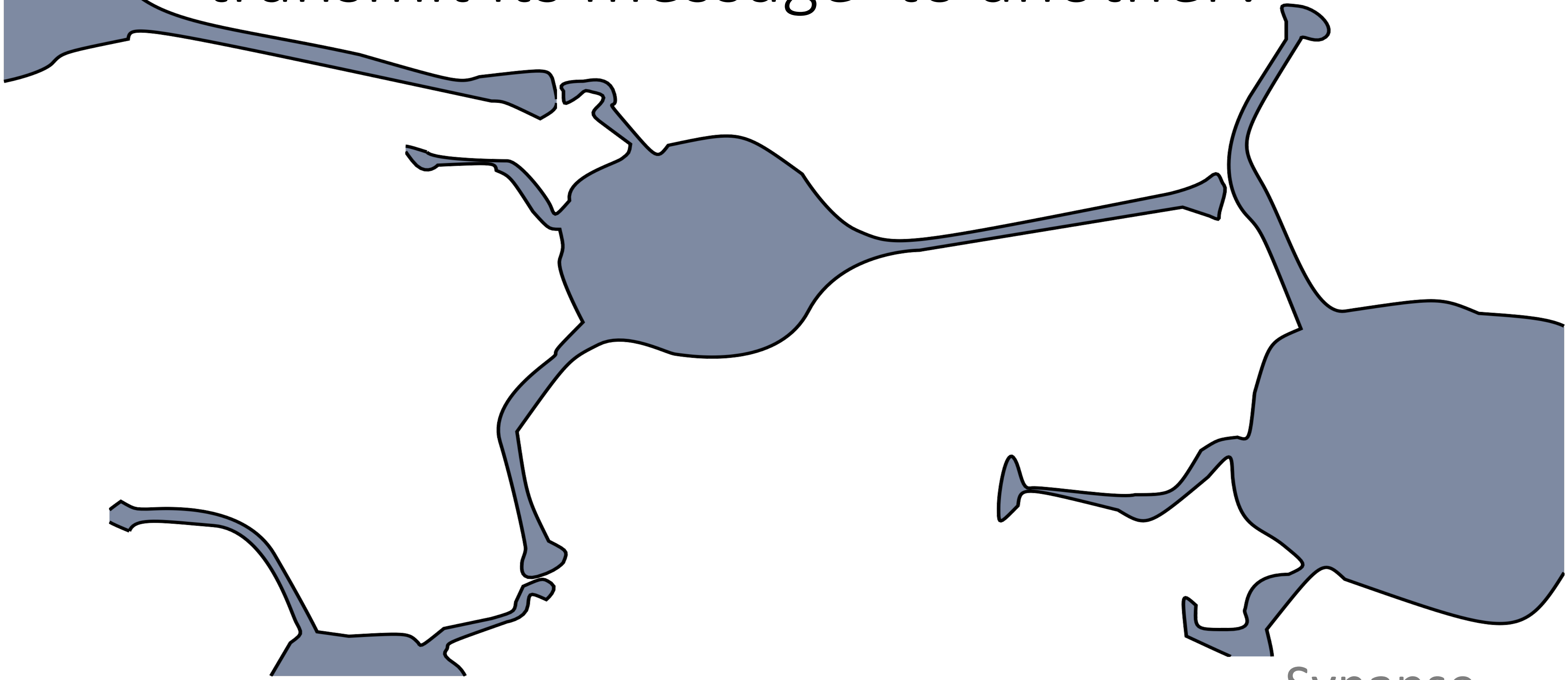
# A virtual neuron

1. Go to: https://phet.colorado.edu/en/simulation/neuron and run the simulation
2. Identify three proteins that we discussed
3. Identify one protein we did not discuss—it is real, but not particularly physiologically relevant for our story of RMPs and APs
4. Describe what's happening at rest (i.e. during resting membrane potential), as regards ionic flow (it helps to zoom in)
5. Describe the order of events when you stimulate the neuron (it helps to slow it down)
6. Turn on the potential chart (bottom right corner). What do you see when you stimulate the neuron? Be specific about voltage changes.
7. Turn on the "Show Concentrations" option (bottom right corner). What do the concentrations tell you at rest? How do those concentrations change when you stimulate the neuron? What do those changes tell you about the effects of an action potential?



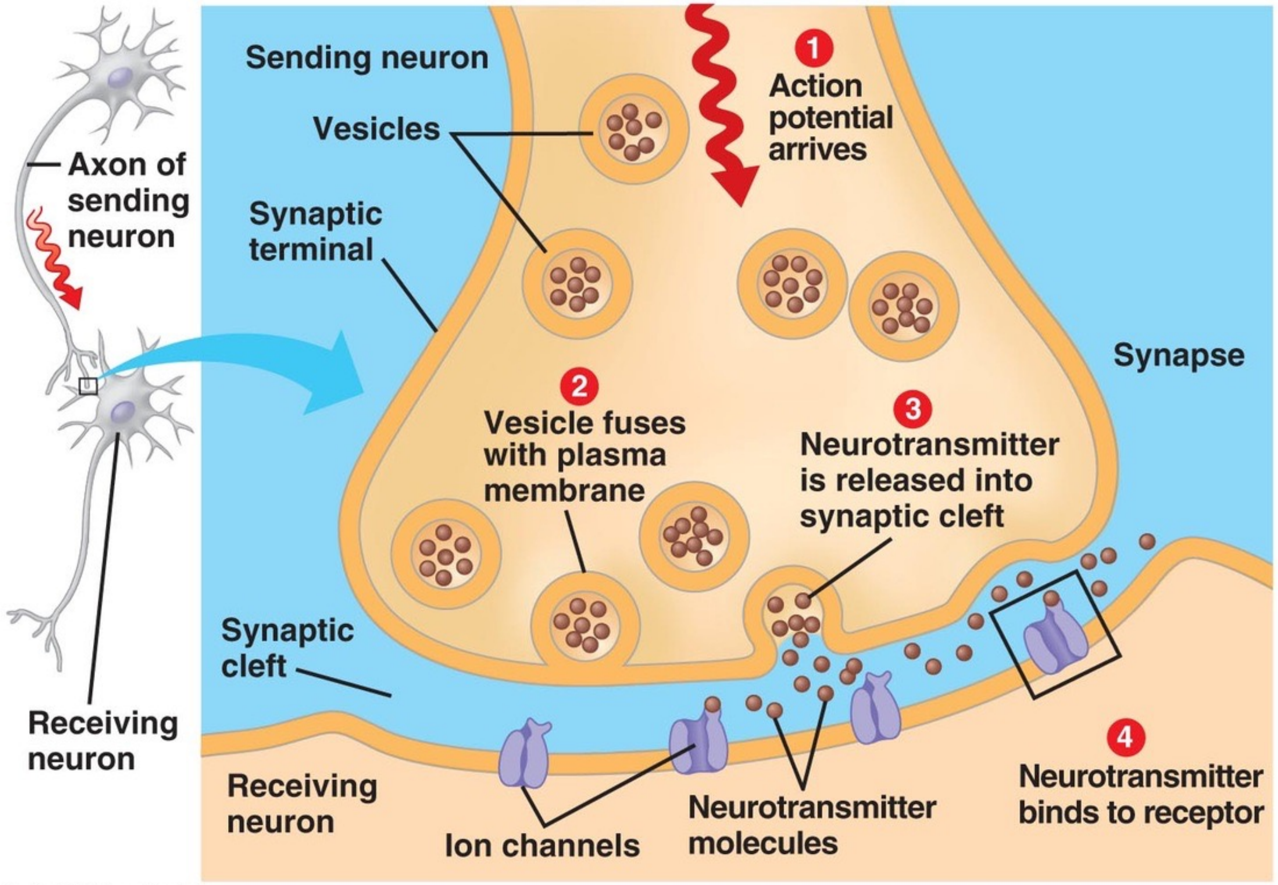

Totally optional extra learning

# Next question: How does one neuron transmit its message to another?



Synapse

# End of the line

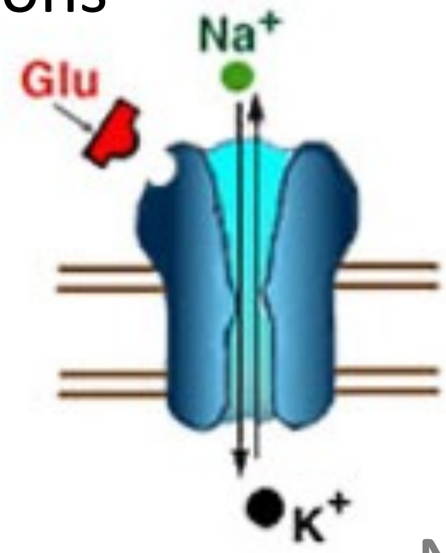


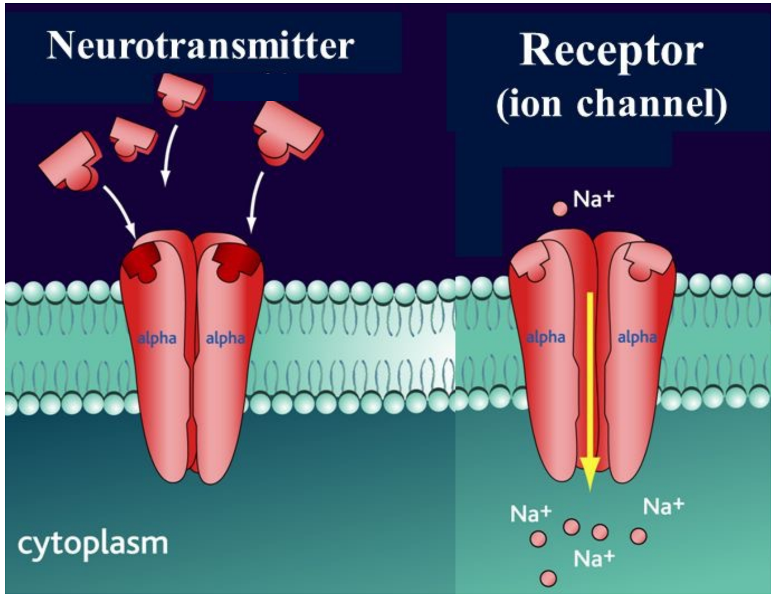



- Axon ends in terminal *boutons* (“buttons”)
- Bouton has *vesicles* (“bubbles”?) filled with *neurotransmitters*
- Action potential travels to bouton and depolarizes it
  - → Causes voltage-gated Ca++ channels to open
  - → Ca++ causes vesicles to fuse with membrane
  - → Neurotransmitters released into synapse

# Welcome to the synapse

- Dendrite membrane has special *receptors* that fit, like lock and key, with the neurotransmitters
- Receptors are often just (closed) channels that open when they bind with neurotransmitter!
    - i.e. *ligand-gated ion channels*
- i.e. EPSPs/IPSPs are just ions crossing the membrane! (Sound familiar?)

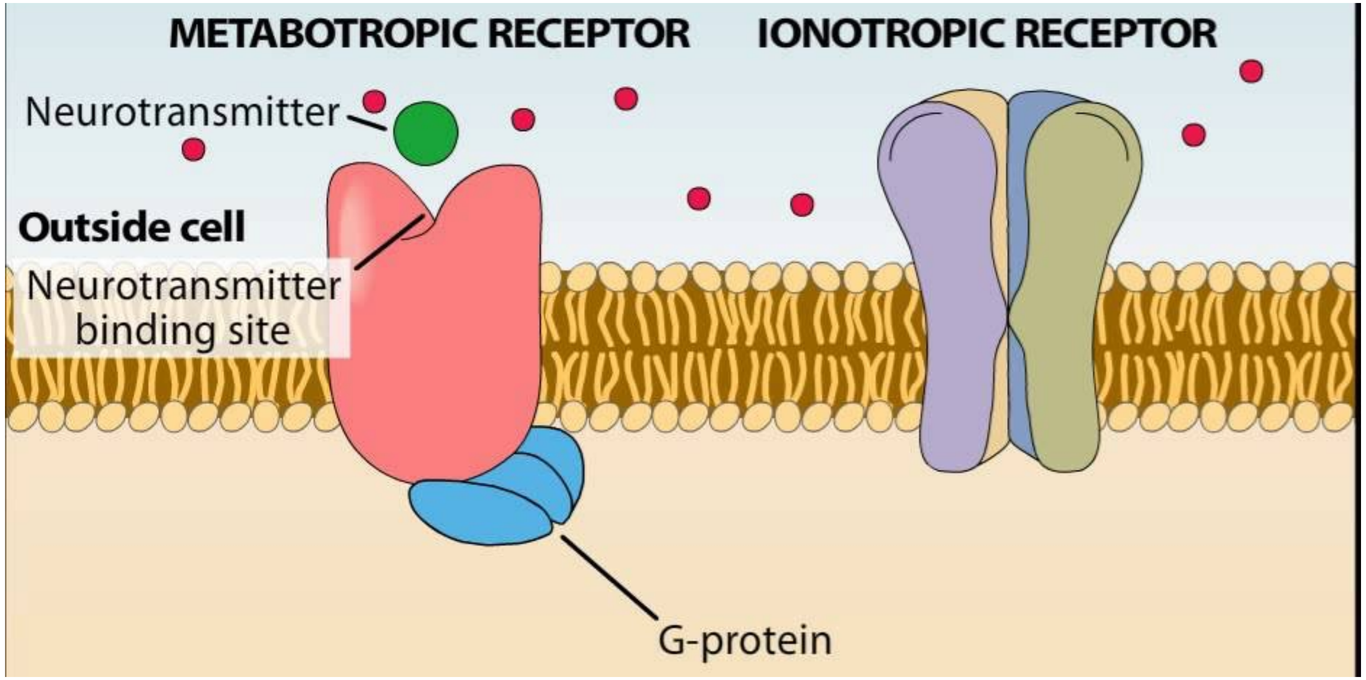



Glu: glutamate, most common excitatory neurotransmitter

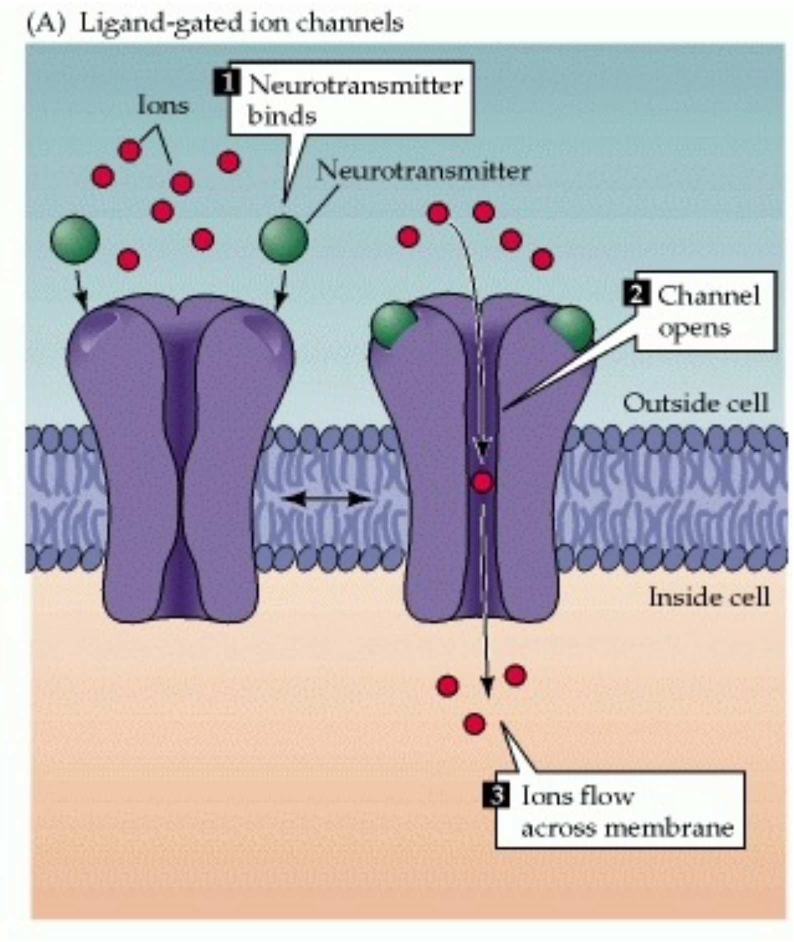
# Receptor types

- Ionotropic (channels)
- Metabotropic (signalling proteins)



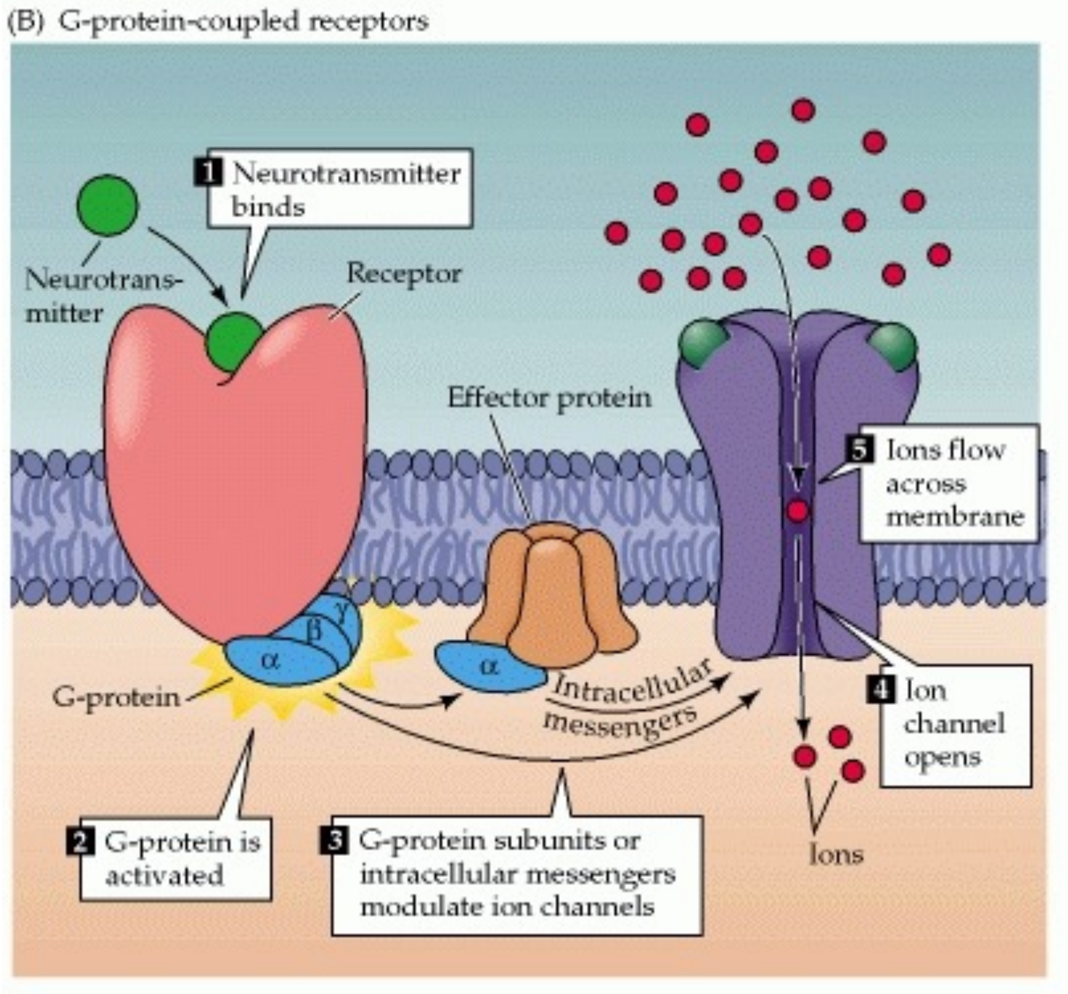
# Receptor types

- Ionotropic
  - AKA ligand-gated ion channels
  - Excitatory (depolarize, EPSPs)
  - Inhibitory (hyperpolarize, IPSPs)
  - Fast, transient effect



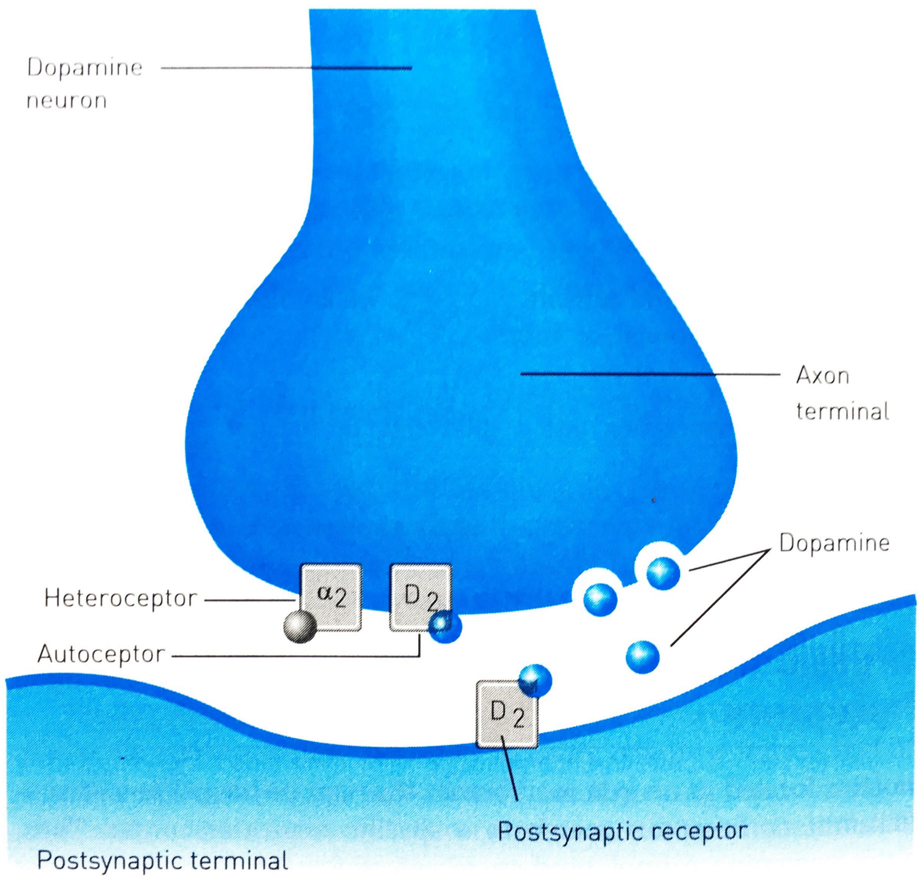
# Receptor types

- Metabotropic
  - AKA G-protein-coupled receptors (GPCRs)
  - Modulate cell
  - Modulate signals
  - Slow, longer lasting effect
  - Cause *signal cascades*



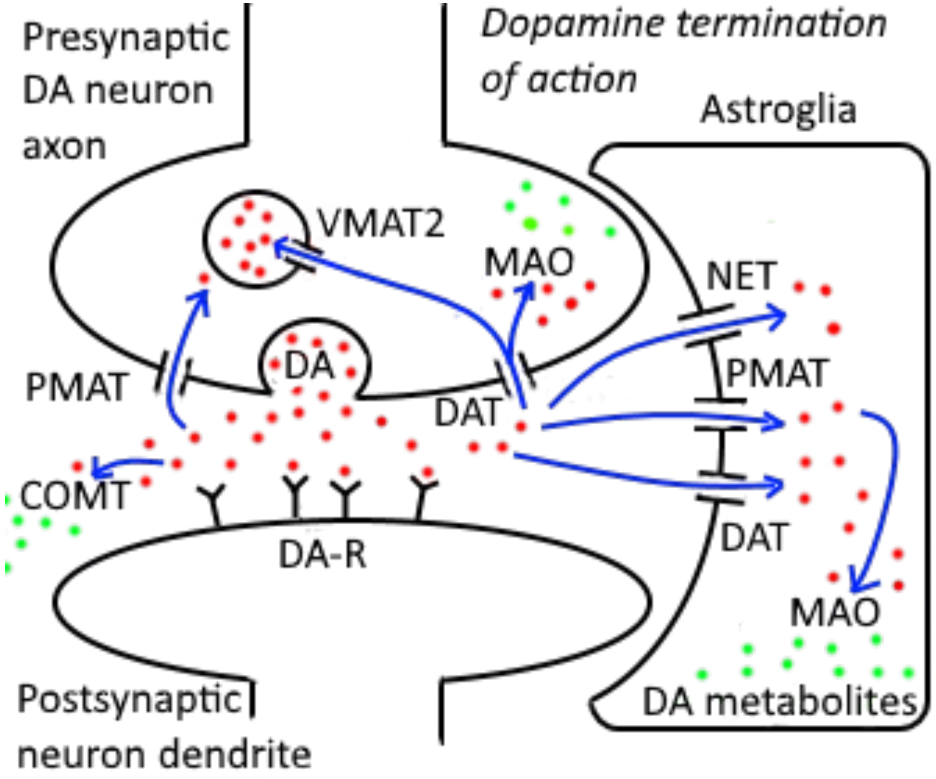
# Receptor locations

- Postsynaptic
- Presynaptic
  - Autoreceptors
  - Heteroreceptors



# Neurotransmitter clean-up

- Diffusion
- Enzymatic degradation
- Re-uptake
  - Pre-synaptic
  - Astrocytes




End of neurotransmission