NEURONS AND SYNAPSES

PG-2

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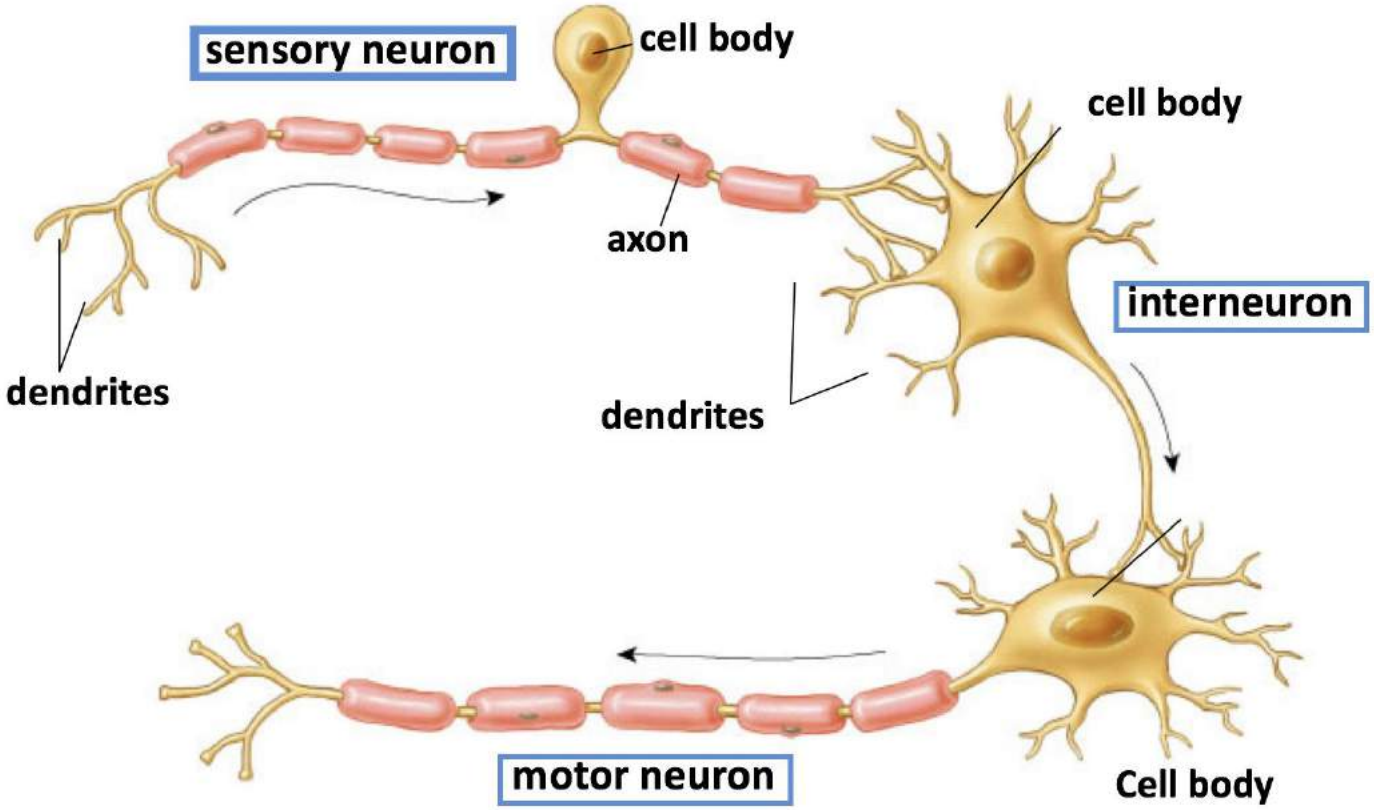
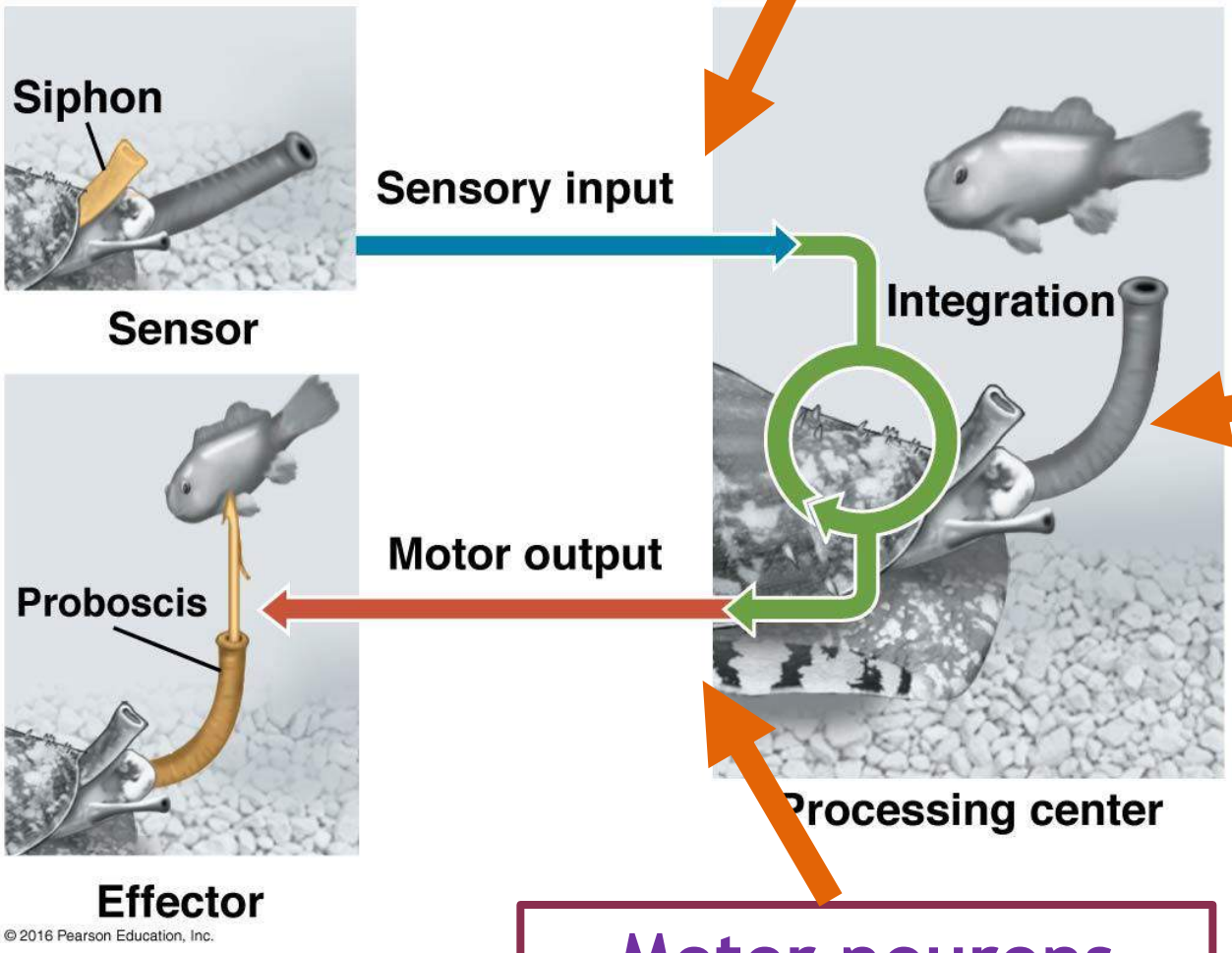
TYPES OF NEURONS

- **Sensory neurons**: info from body sensors → spine and brain (central nervous system (CNS))
- **Interneurons**: connect sensory + motor neurons (peripheral nervous system (PNS)), or local connections between brain + spinal cord (CNS)
- **Motor neurons**: CNS → body (effectors = muscles, glands)



Sensory neurons

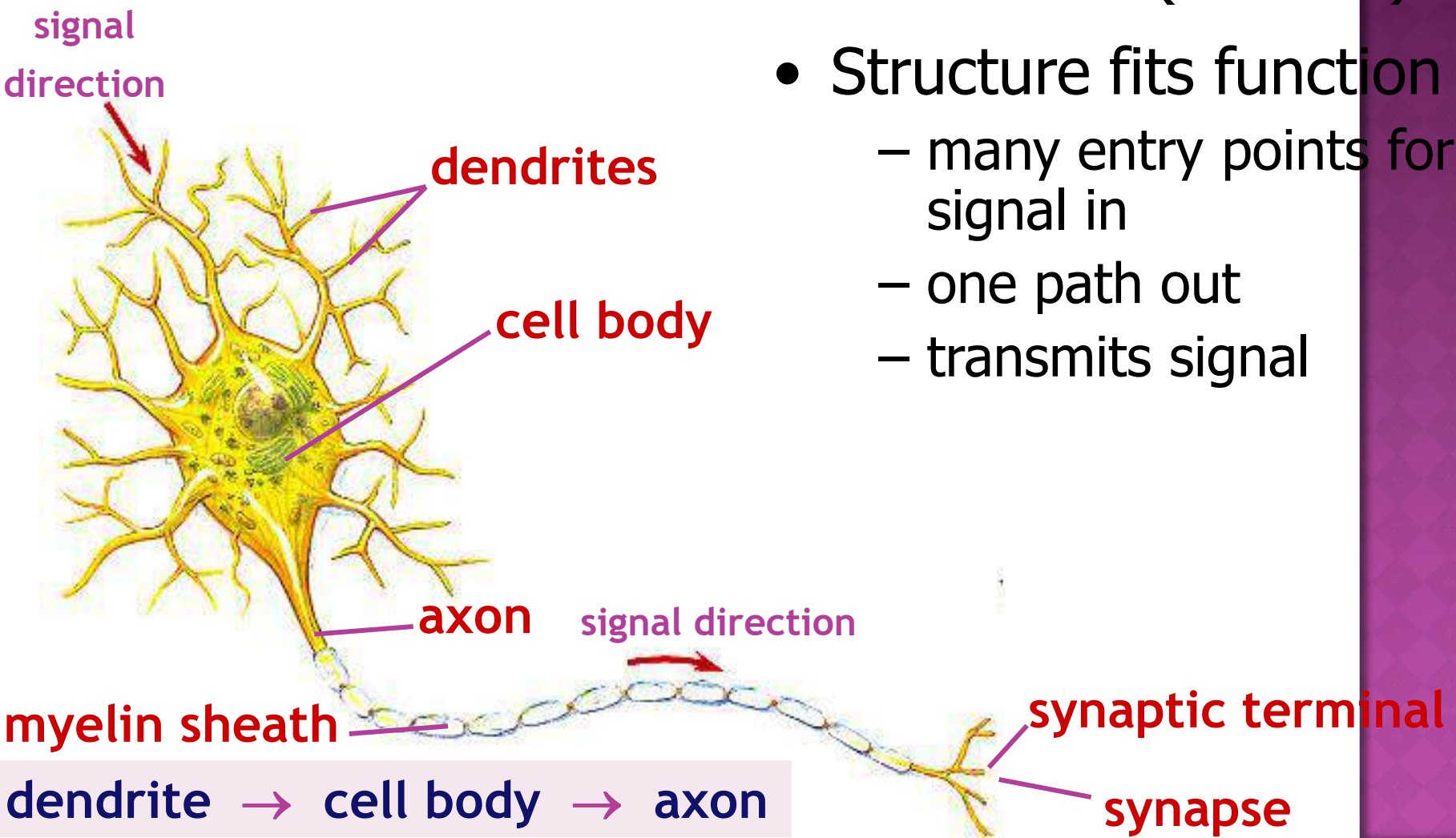


Interneurons

Motor neurons

NERVE CELLS

- Nerve cell (neuron)
- Structure fits function
  - many entry points for signal in
  - one path out
  - transmits signal



dendrite → cell body → axon

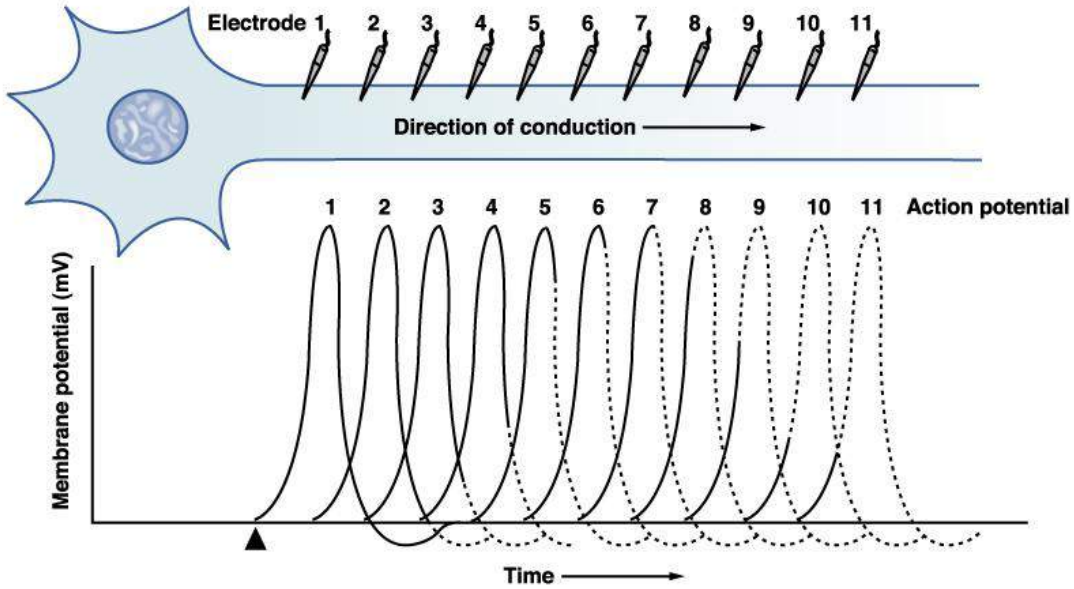
TRANSMISSION OF A SIGNAL FROM NERVE CELL TO NERVE CELL

Think dominoes!

- **start the signal**
  - knock down line of dominoes by tipping 1st one
    → trigger the signal
- **propagate the signal**
  - do dominoes move down the line?
    → no, just a wave through them!
- **re-set the system**
  - before you can do it again,
    have to set up dominoes again
    → reset the axon

TRANSMISSION OF A NERVE SIGNAL

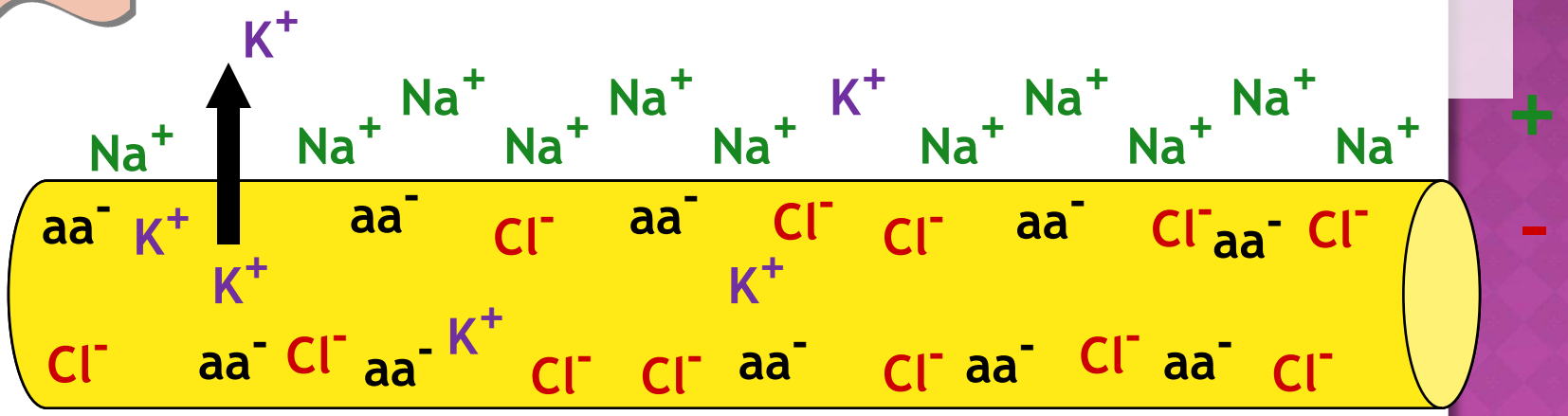
- Neuron has a similar system
  - protein channels are set up in the neuron's cell membrane
  - once the first channel is opened, the rest open in succession
    - all or nothing response
  - a "wave" action travels along neuron
  - The channels have to re-set so a neuron can react again



CELLS: SURROUNDED BY CHARGED IONS

- Cells live in a sea of charged ions
  - anions (negative)
    - more concentrated within the cell
    - Cl^-, charged amino acids (aa^-)
  - cations (positive)
    - more concentrated in the extracellular fluid
    - Na^+

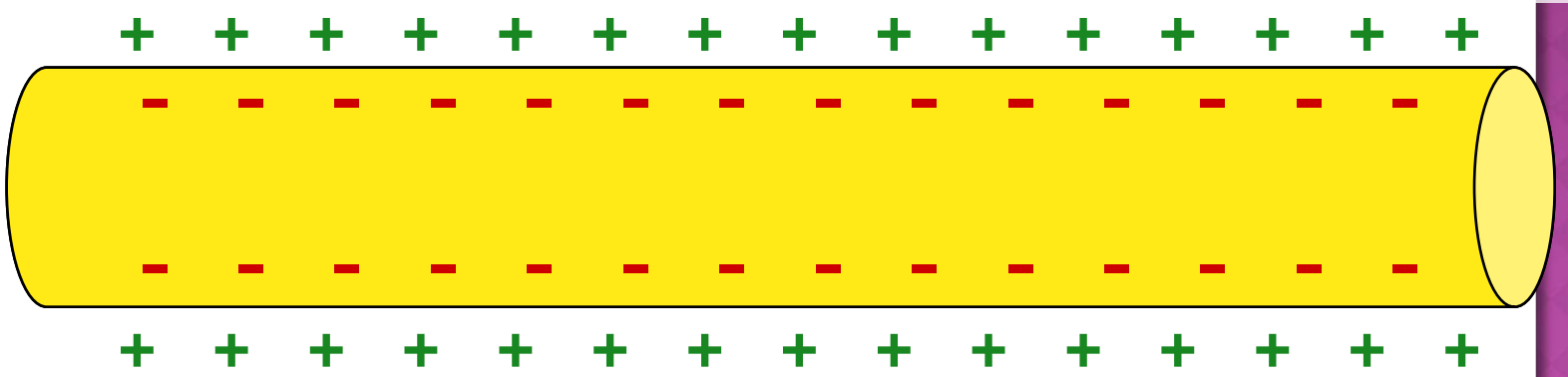
channel leaks K^+



1. RESTING POTENTIAL

Cells have voltage!

- Opposite charges on opposite sides of cell membrane
  - membrane is polarized
    - negative inside; positive outside
    - charge gradient
    - stored energy (like a battery)

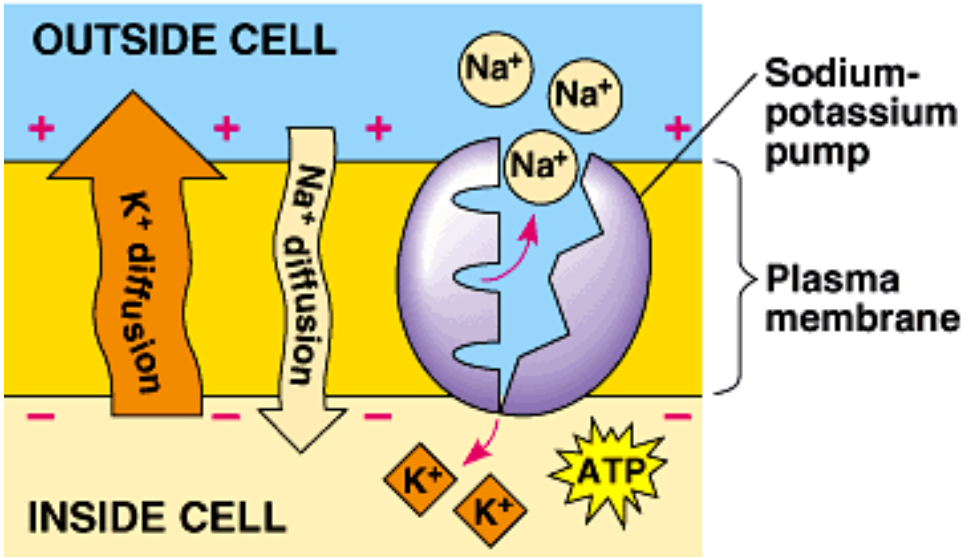


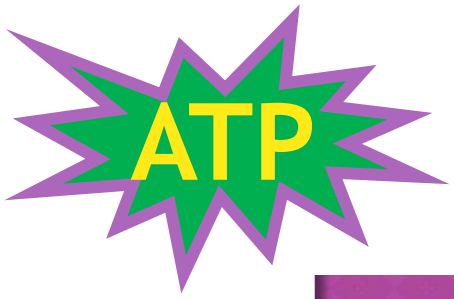
1. RESTING POTENTIAL

Sodium-Potassium pump

Active transport using a protein in membrane (requires ATP)

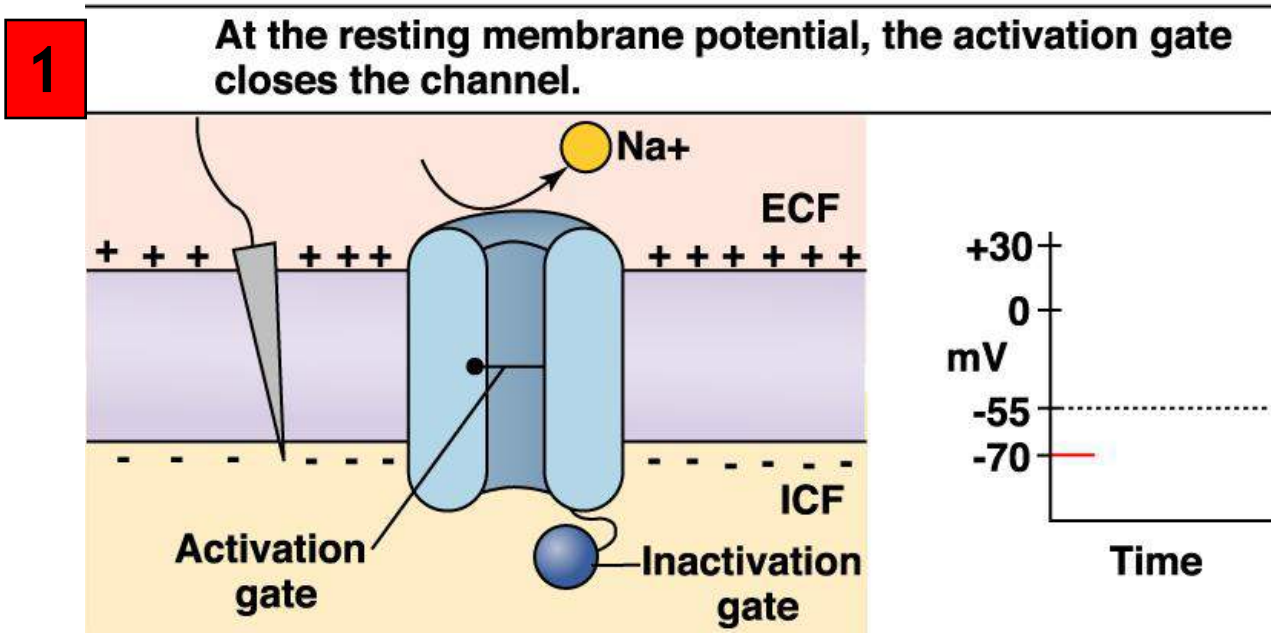
The Protein pumps out 3 Na^+ and pumps in 2 K^+ in across membrane. 3 positives out and 2 positives in is what creates the negative charge inside and the positive outside the cell





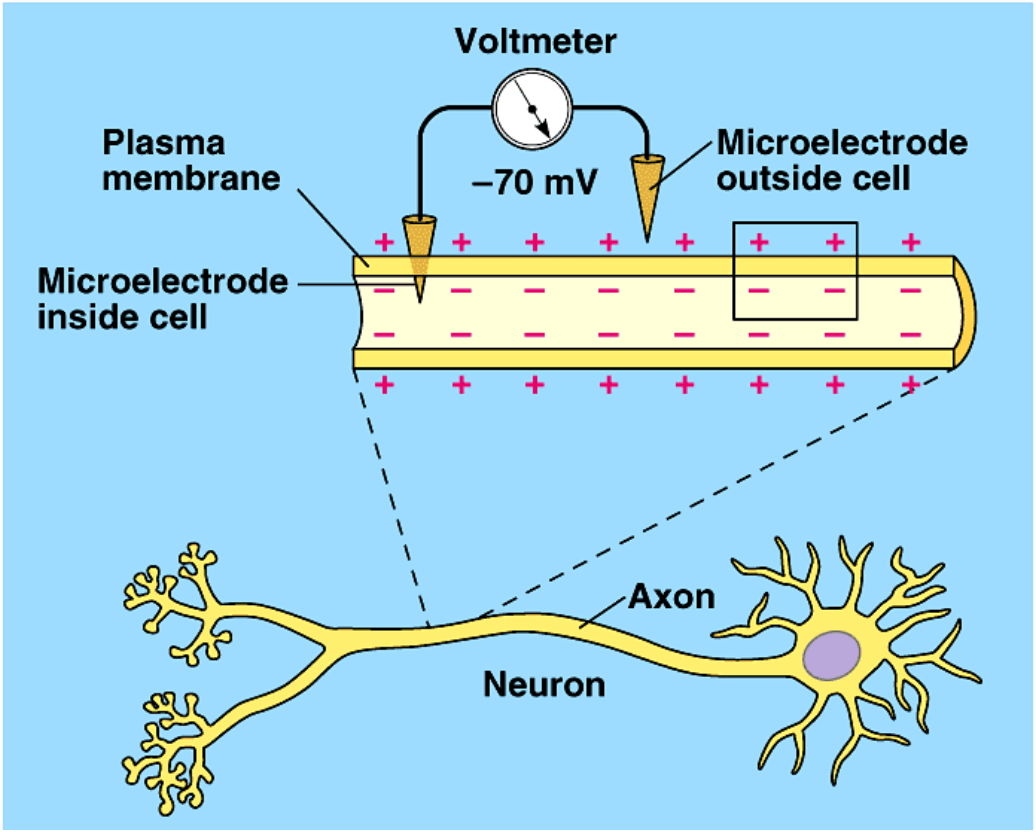
1. RESTING POTENTIAL

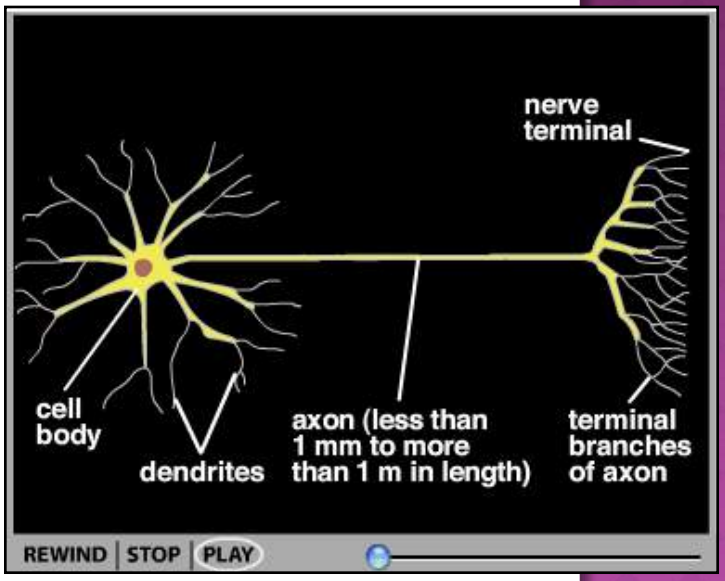
- Neurons are also highly polarized (at about –70mV) due to:
  - Differential membrane permeability to K^+ and Na^+
  - The electrogenic nature of the Na^+/K^+ pump
  - The presence of intracellular impermeable anions



1. Resting Potential

MEASURING CELL VOLTAGE





unstimulated neuron = resting potential of -70mV

2. Stimulus (threshold potential)

- Changes in mV must reach at least -55mV (for an all or nothing reaction) for a true action potential to kick in. This is a intercellular communication that cause Na+ ions to rush in.

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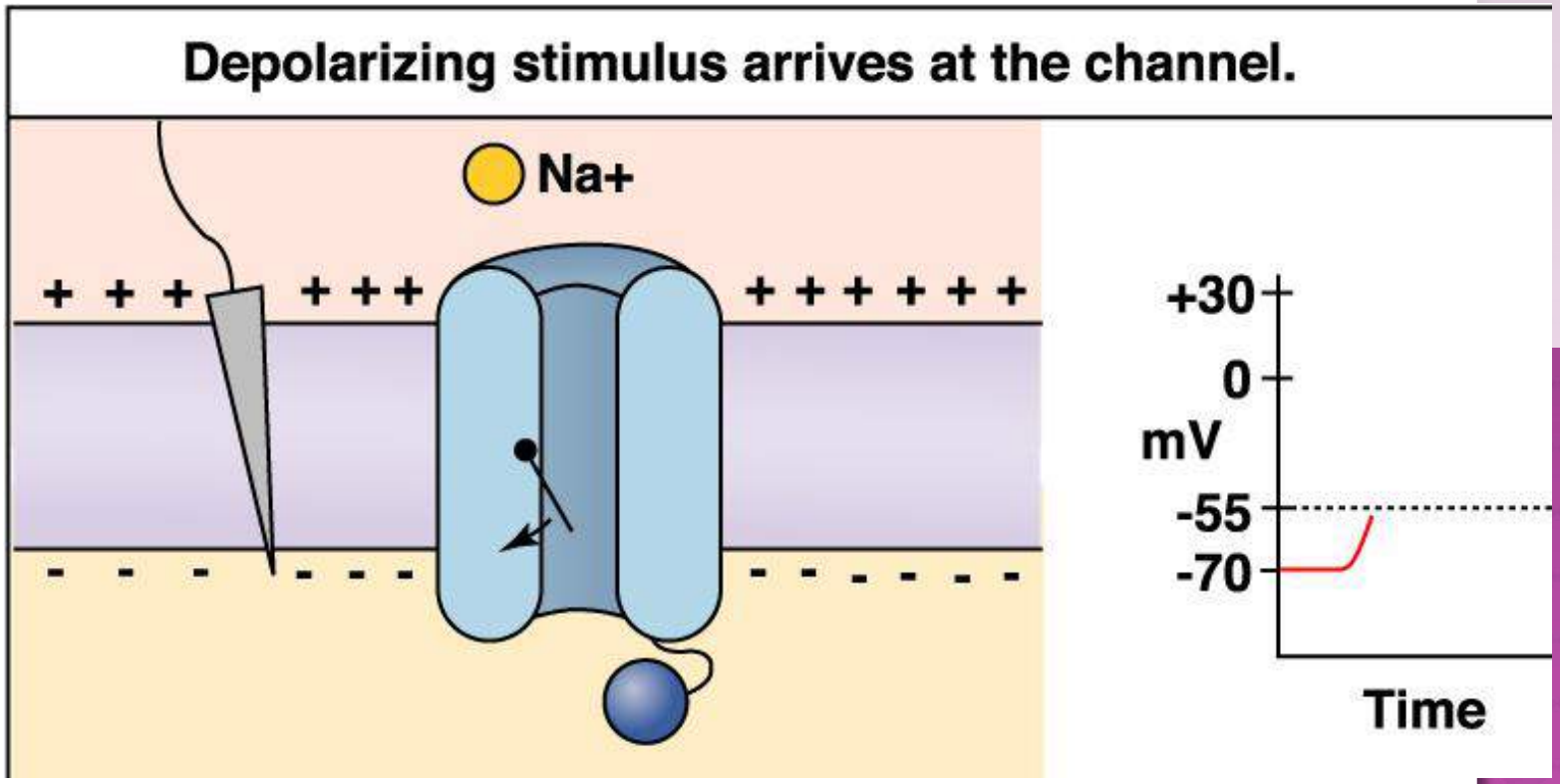
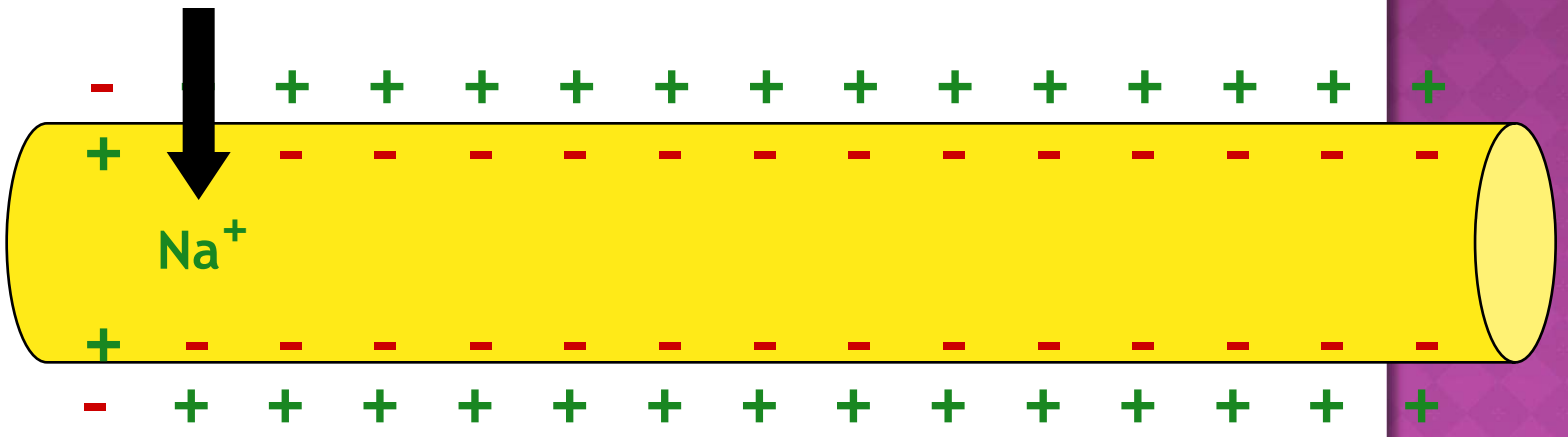


2. Stimulus (threshold potential)

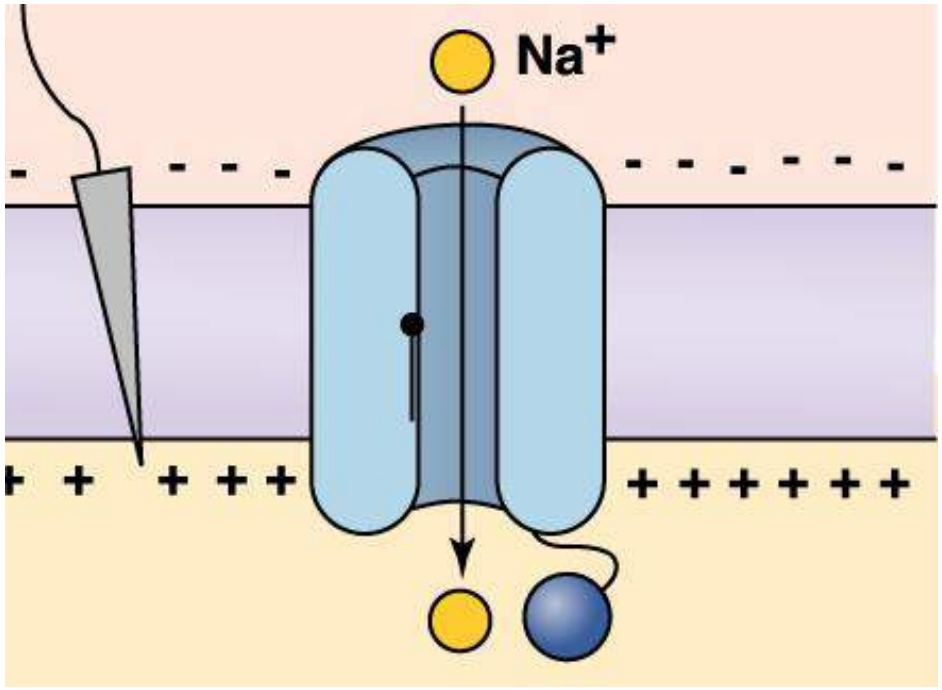
- Stimulus: nerve is stimulated (allows for opening of some Na+ channels)
  - If enough Na+ enters and the neuron reaches a threshold potential of -55mV
    - Opens many Na^+ channels in that portion of the cell membrane
    - **Na^+ ions diffuse into the cell**
  - charges reverse at that point on neuron
    - positive inside; negative outside
    - cell becomes depolarized

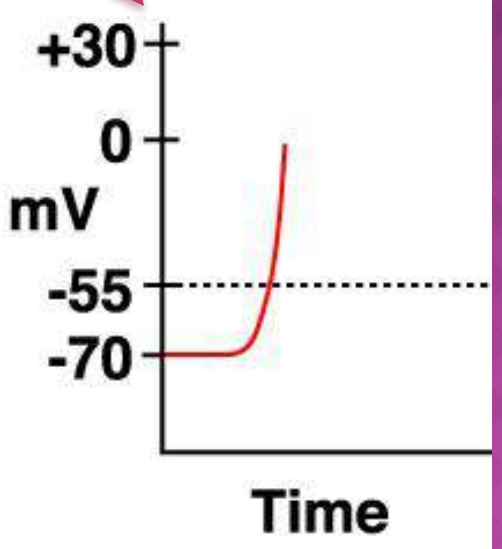


3. Depolarization (Voltage-gated channels)

This all or nothing reaction triggers many Na+ Voltage-gated channels to open along the nerve cell. This causes the cell to quickly have a **reversal of charge inside the cell**

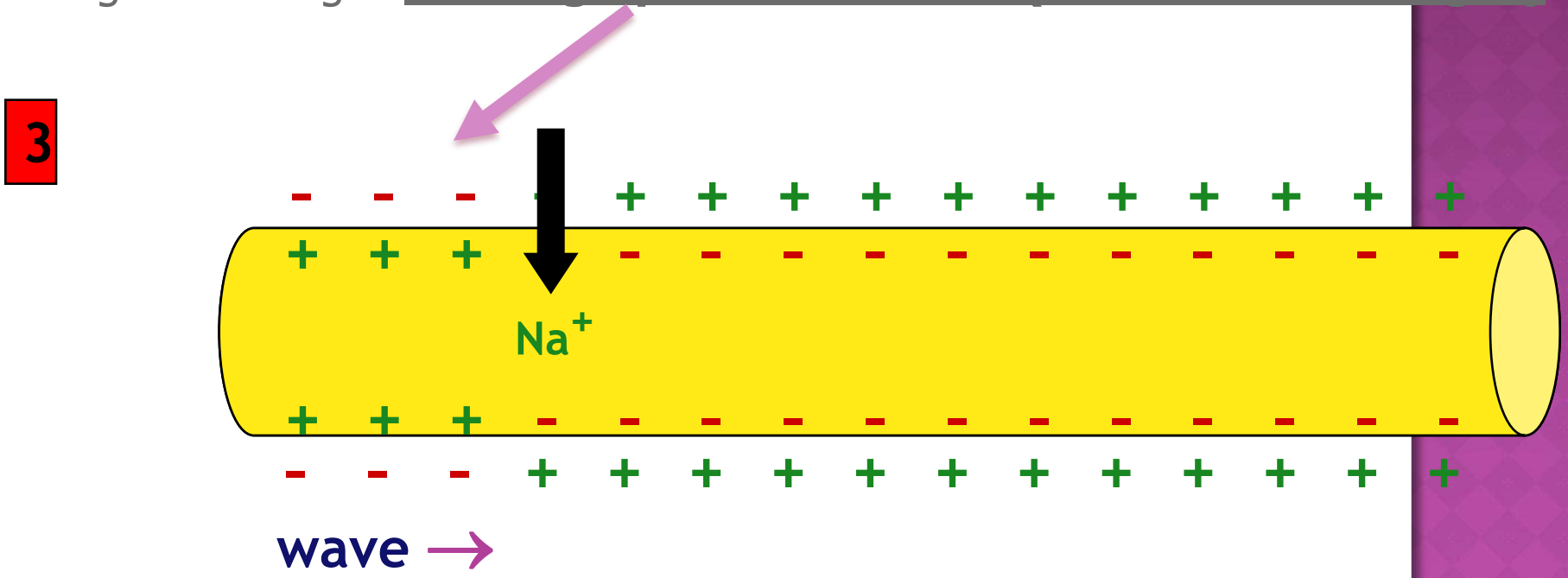
With activation gate open, Na^+ enters the cell.





3. Depolarization (Voltage-gated channels)

- The wave (chain reaction): once the stimulus has occurred a nerve impulse travels down neuron
- A chance in the charge inside the cell allow Na^+ diffuse into the cell through the open "voltage-gated" channels **down its concentration gradient**.
  - Na^+ ions continue to diffuse into cell as the "wave" moves down neuron creating a positive charge build up on the inside of the nerve cell, with negative charges **building up on the outside (a reversal of charges)**



4. Action Potential

- This reversal of charge from -70mV to as much a +40mV is **action potential** (a brief reversal of charge or a depolarization).

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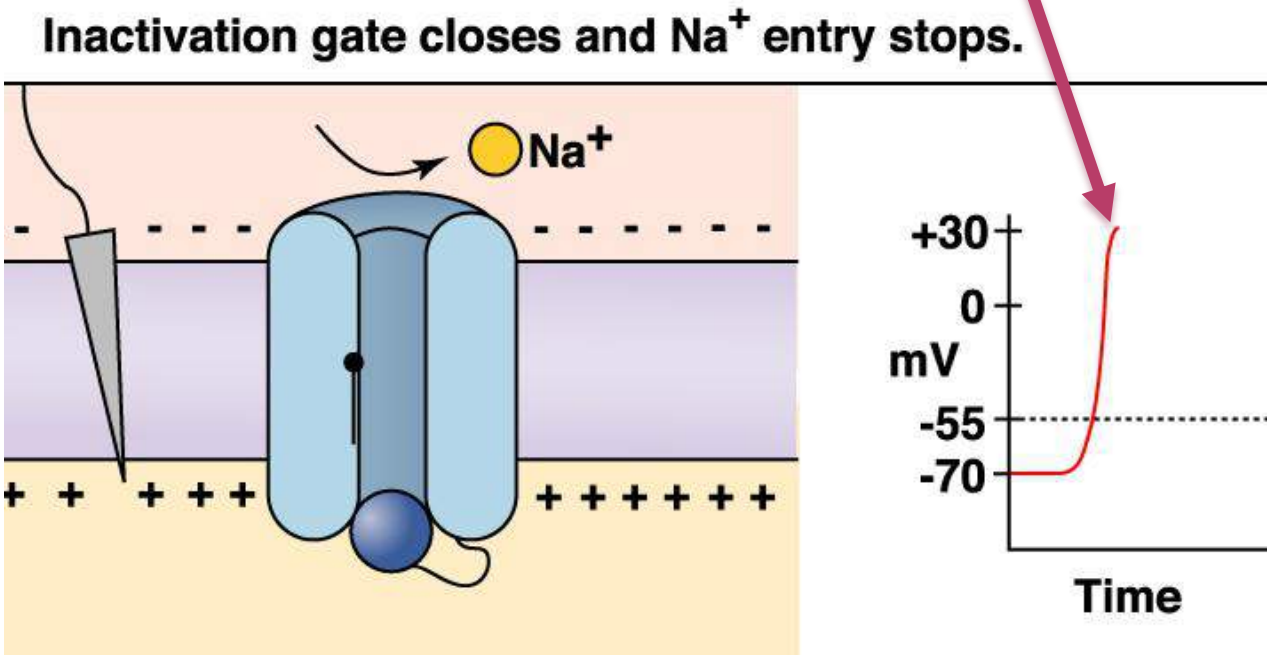
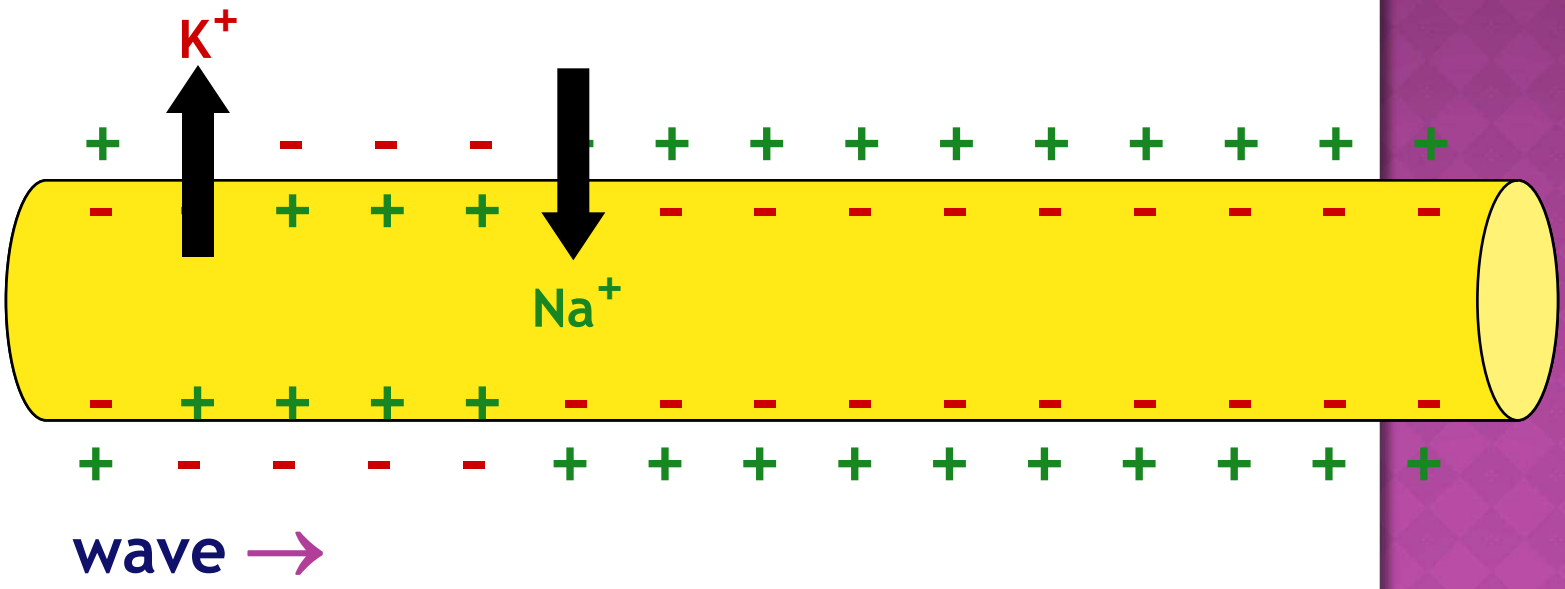


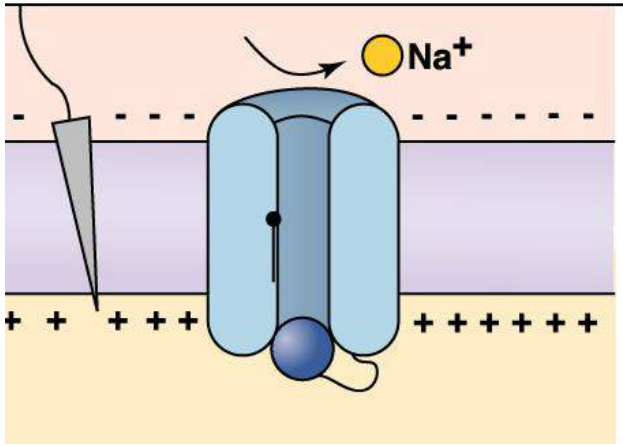
5. REPOLARIZATION

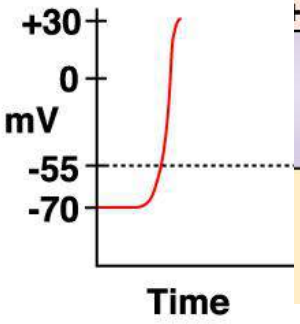
- Re-set: 2nd wave travels down neuron
  - K^+ "voltage-gated" channels open (with a slight delay)
    - K^+ channels open up more slowly than Na^+ channels
  - K^+ ions diffuse **out** of cell, while the **Na+ channels close behind the action potential**
  - charges reverse back at that point
    - negative inside; positive outside



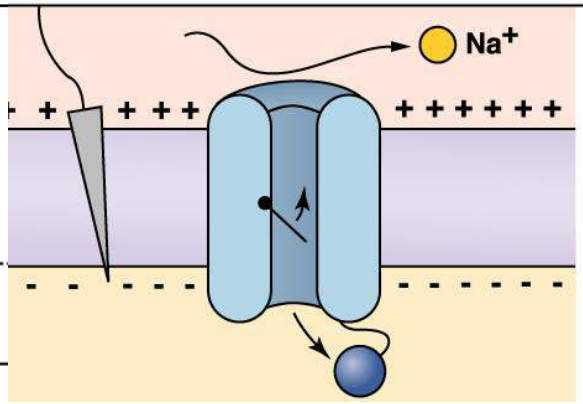
5. REPOLARIZATION

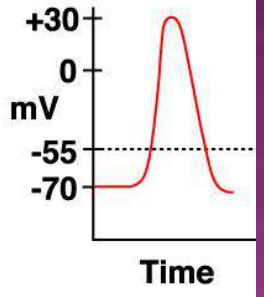
Inactivation gate closes and Na^+ entry stops.

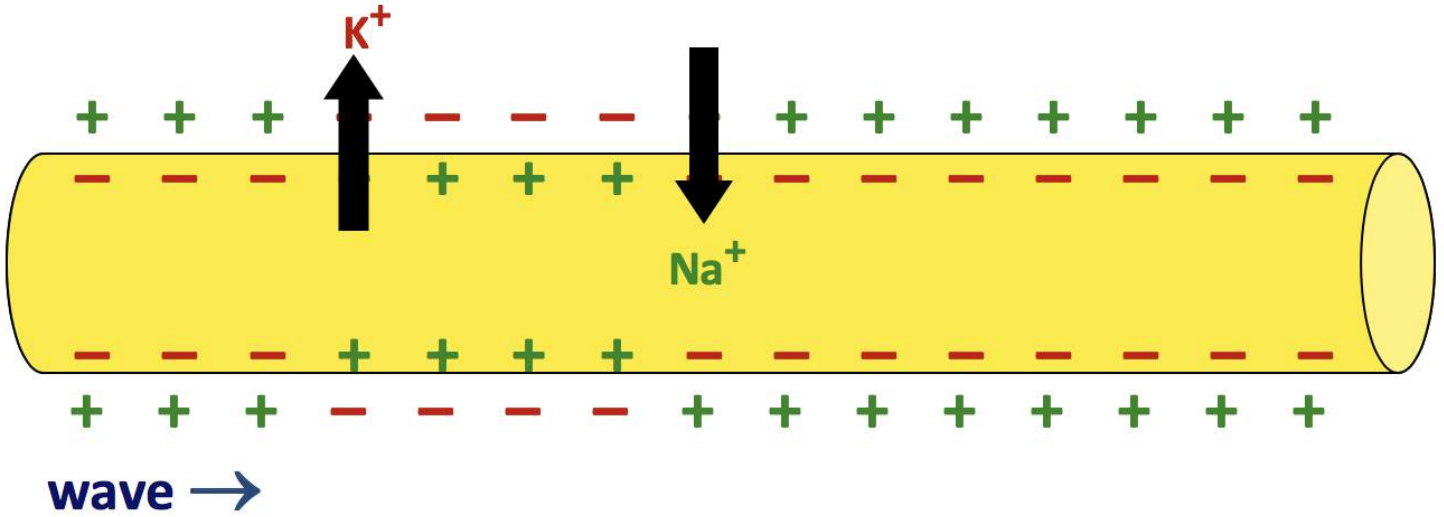




During repolarization caused by K^+ leaving the cell, the two gates reset to their original positions.

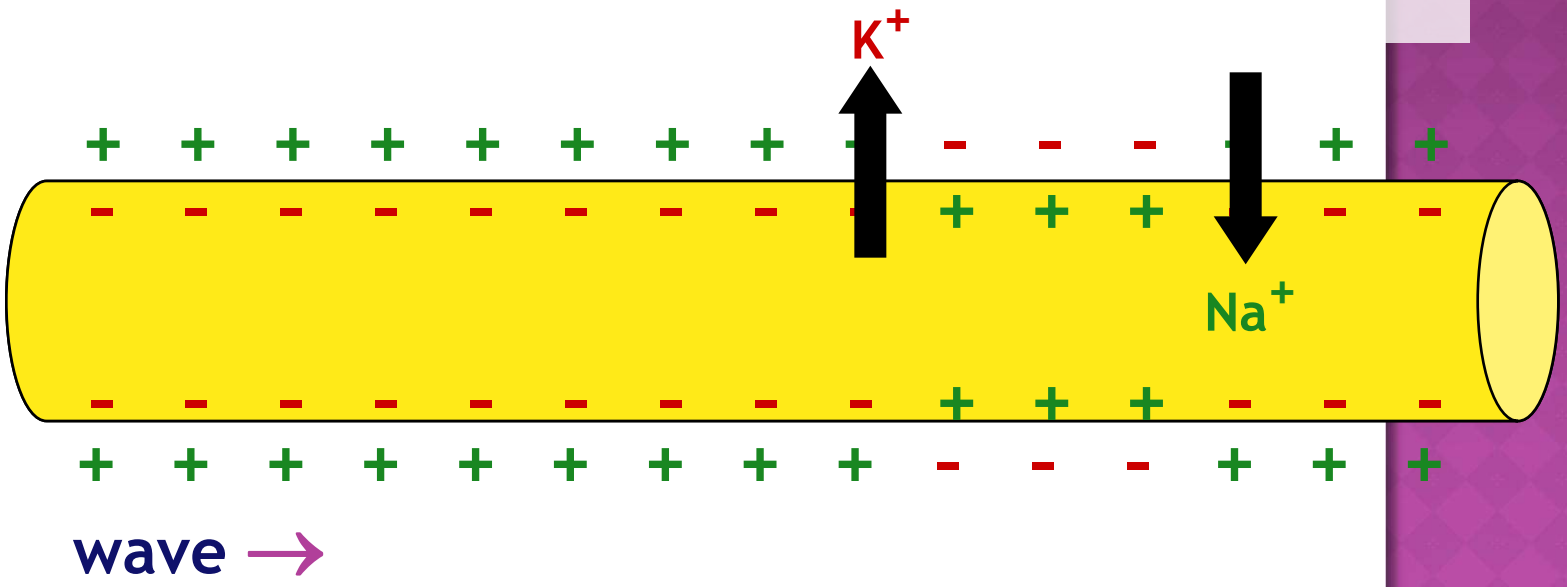






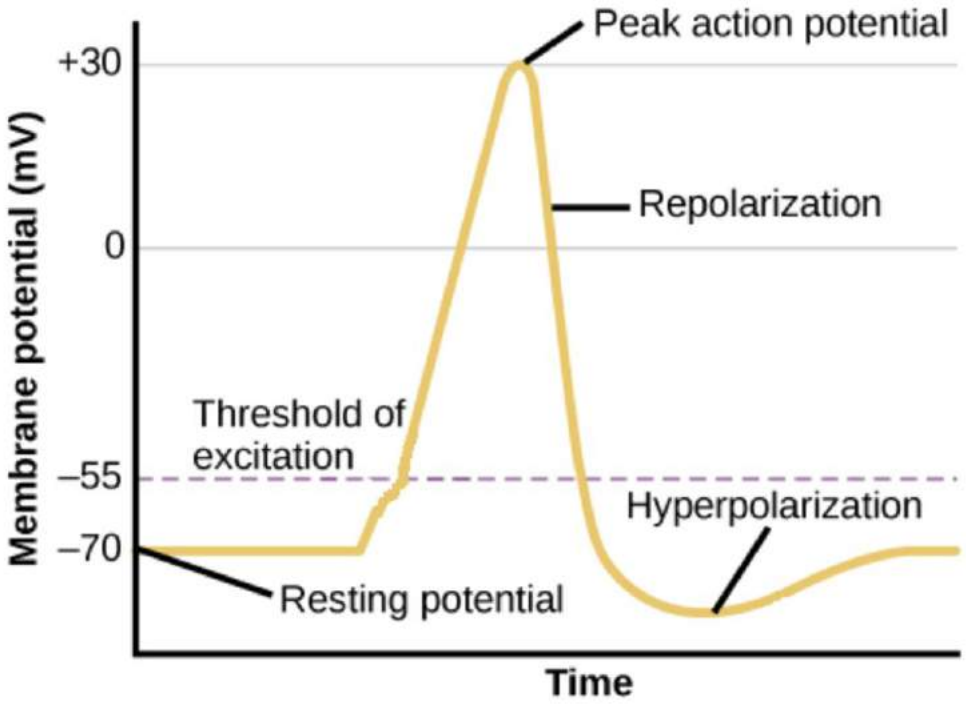
5. Repolarization

- Ion channels open & close in response to changes in charge across membrane
  - Na^+ channels open quickly in response to depolarization & close slowly
  - K^+ channels open slowly in response to depolarization & close slowly



6. Hyperpolarization- ensures one way direction of signal

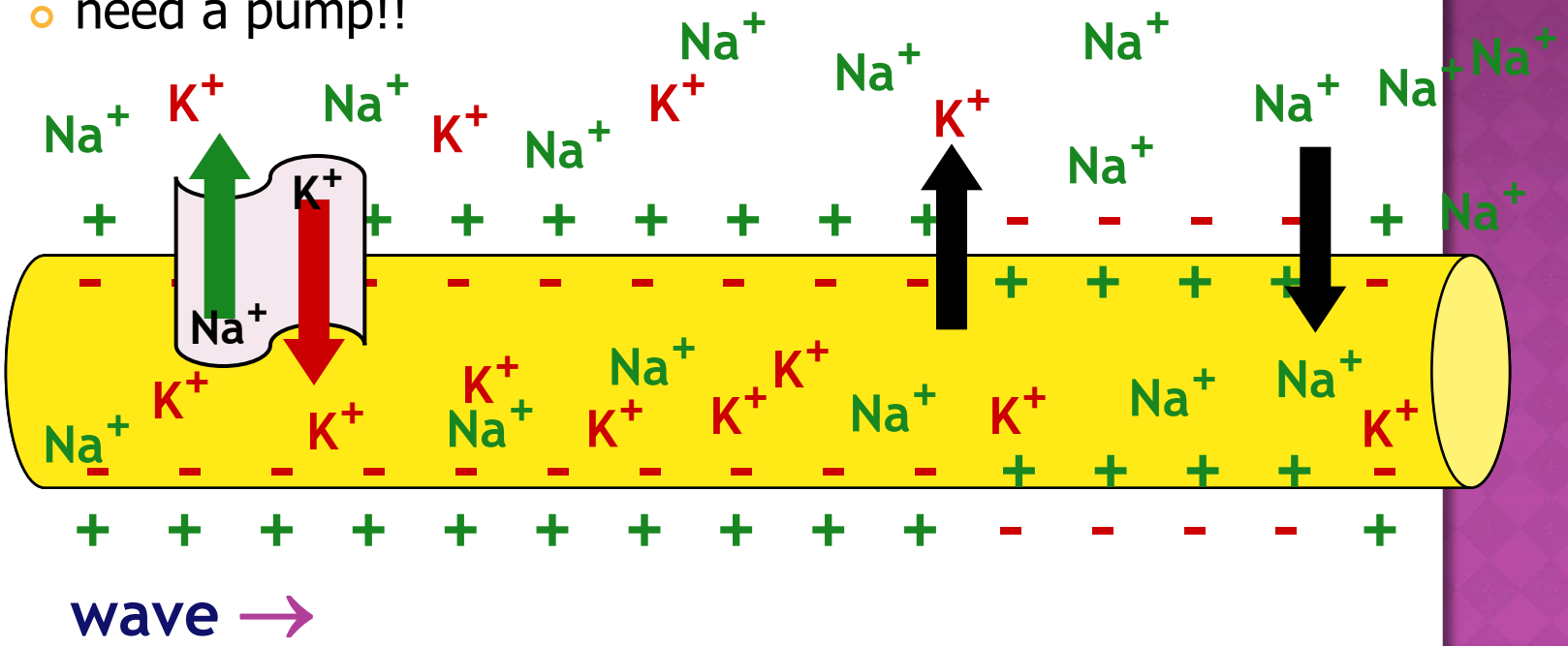
6. <u>Undershoot</u> (Hyperpolarization) Potassium Voltage-gated channels open and Sodium voltage-gated channels close. For a short period of time this cause a change in the movement of ions. This change result in a temporary charge lower negative change then resting potential, before settling back to resting potential.



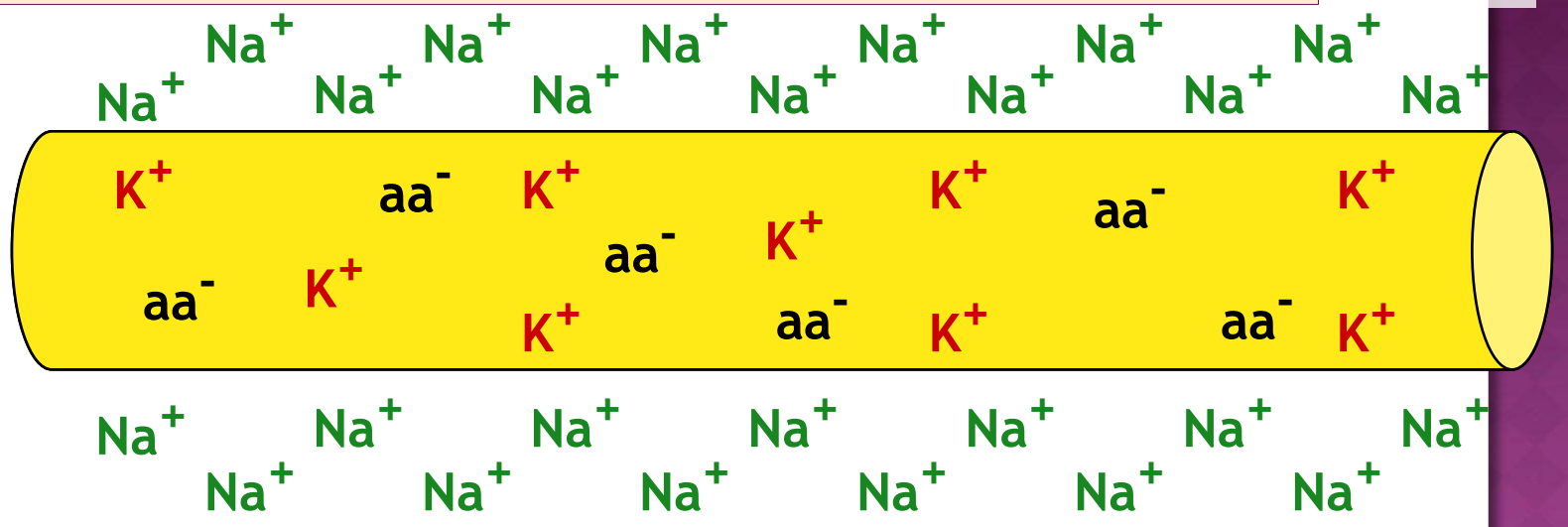
5. Repolarization

HOW DOES THE NERVE RE-SET ITSELF?

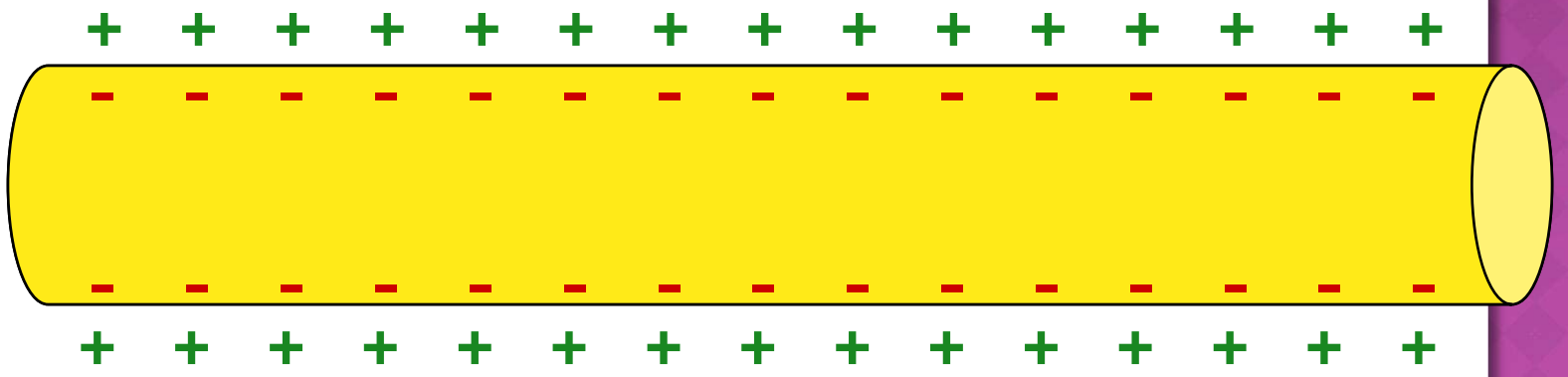
- After firing a neuron has to re-set itself
  - Na^+ needs to move back out
  - K^+ needs to move back in
  - both are moving against concentration gradients
    - need a pump!!



NEURON IS READY TO FIRE AGAIN

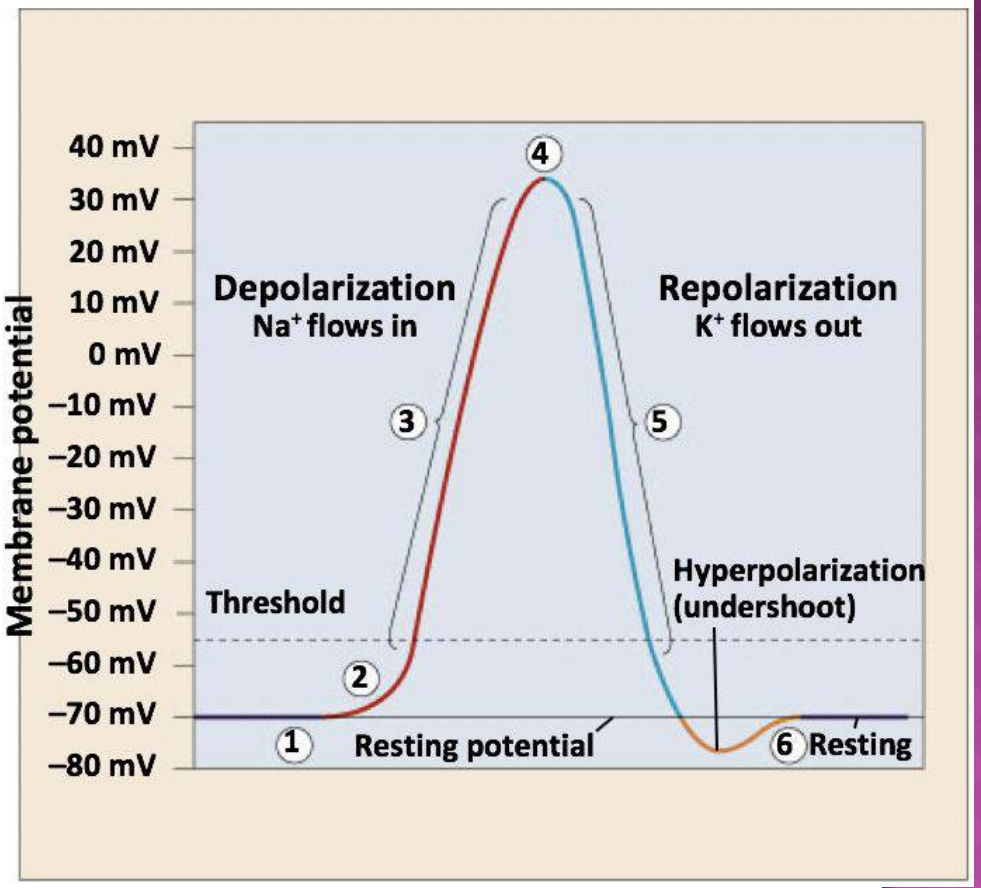


resting potential



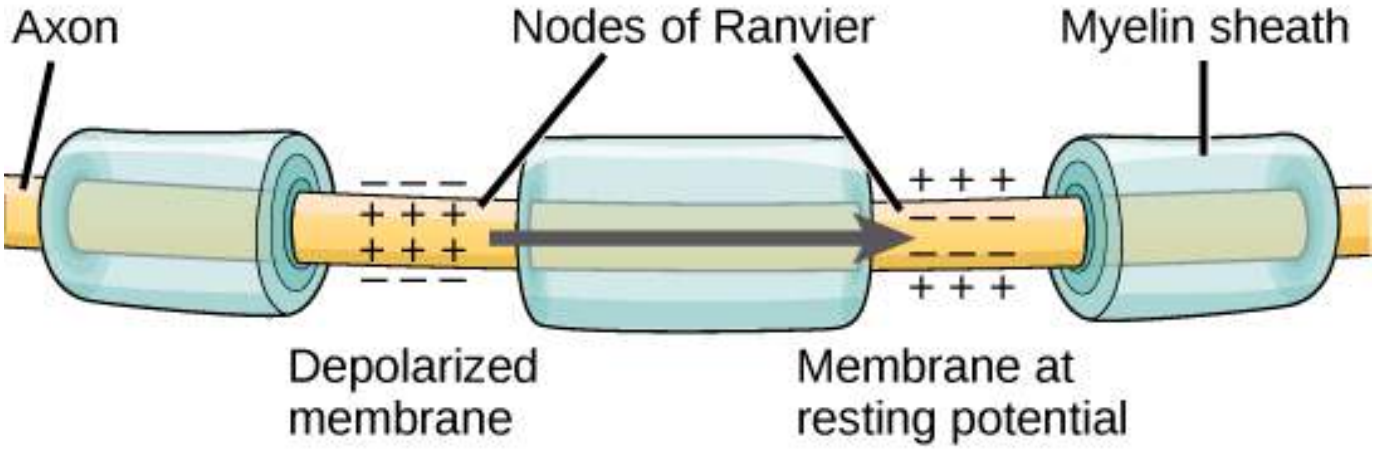
ACTION POTENTIAL GRAPH

1. Resting potential
2. Stimulus reaches threshold potential
3. Depolarization
   Na^+ channels open;
   K^+ channels closed
4. Action Potential
   Na^+ channels close; K^+ channels open
5. Repolarization
   reset charge gradient
6. Undershoot
   K^+ channels close slowly



Myelination Conduction

- As **myelin acts as an insulator** myelinated axons only allow **action potentials to occur at the unmyelinated nodes of Ranvier**.
- The result of this is that the impulse travels much more quickly (up to 200 m/s) along myelinated axons compared to unmyelinated axons (2 m/s).
- The **myelin sheath also reduces energy expenditure** over the axon as the quantity of sodium and potassium ions that need to be pumped to restore resting potential is less than that of a un-myelintated axon

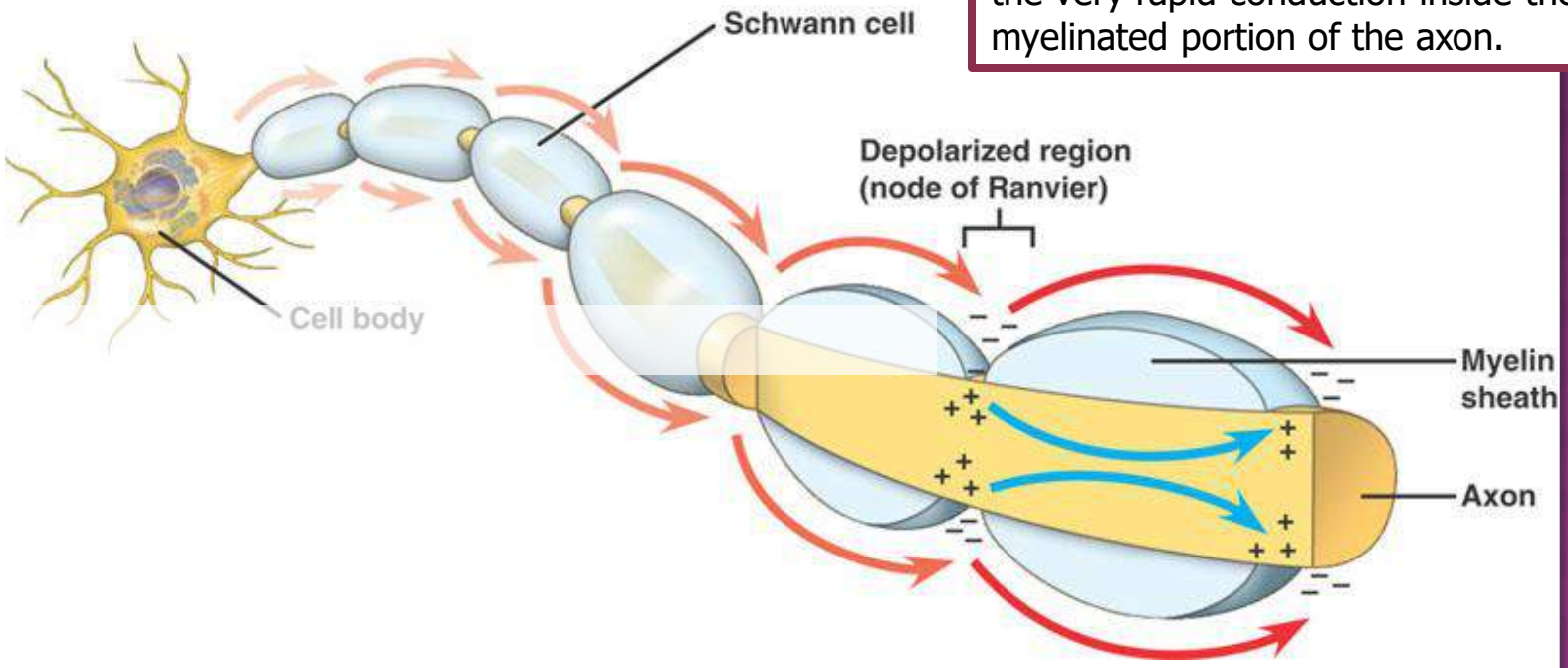


*The jump along the axon is actually just the very rapid conduction inside the myelinated portion of the axon.

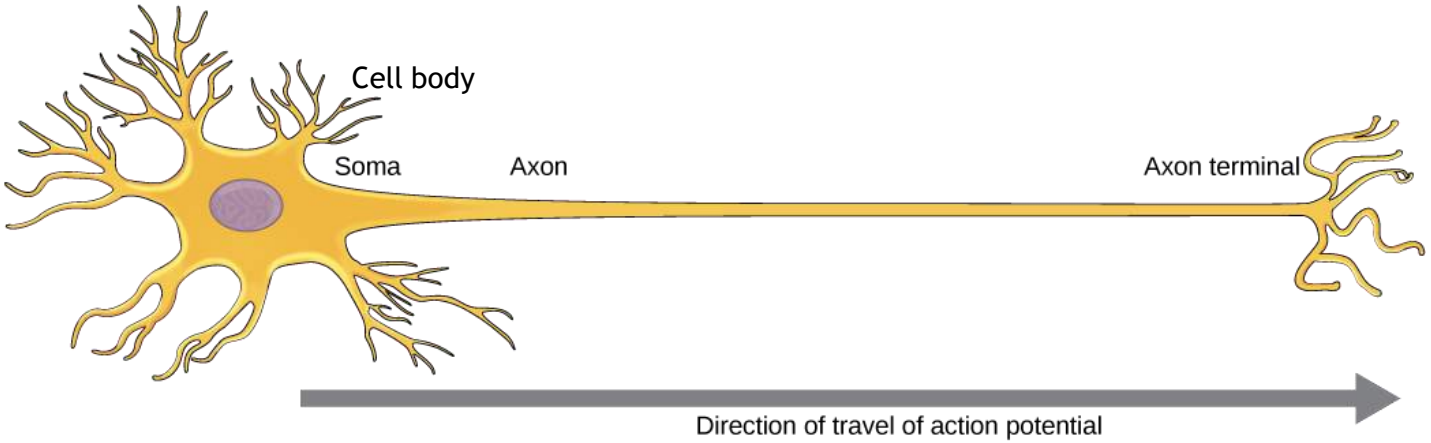
Saltatory Conduction

- This forces the action potential to jump* from node to node (saltatory conduction).
- Saltatory conduction from node to node also reduces degradation of the impulse and hence allows the impulse to travel longer distances than impulses in unmyelinated axons.

*The jump along the axon is actually just the very rapid conduction inside the myelinated portion of the axon.



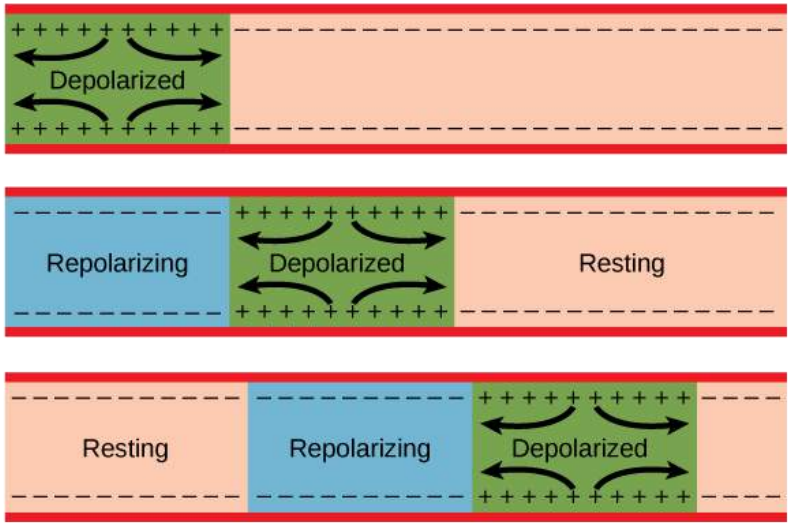
Propagation of a nerve impulse in un-myelinated axons

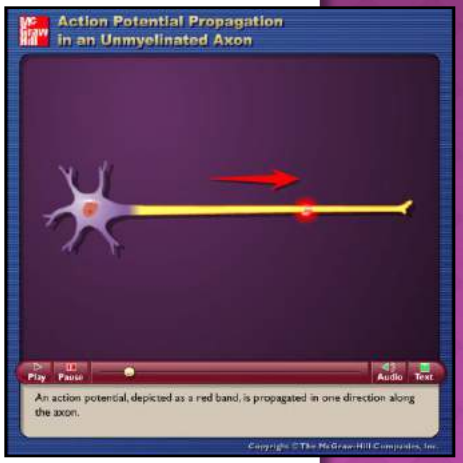


a. In response to a signal, the soma end of the axon becomes depolarized.

b. The depolarization spreads down the axon. Meanwhile, the first part of the membrane repolarizes. Because Na^+ channels are inactivated and additional K^+ channels have opened, the membrane cannot depolarize again.

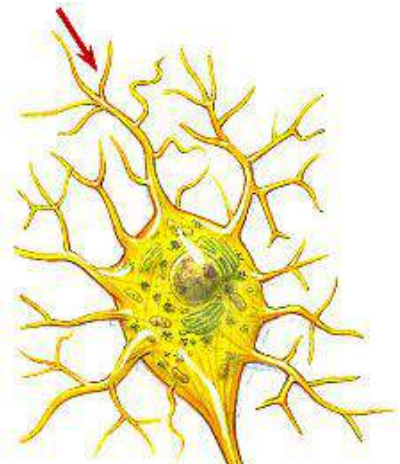
c. The action potential continues to travel down the axon.





http://highered.mheducation.com/olc/dl/120107/bio_d.swf

What happens at the end of the axon?



Impulse has to jump the synapse!

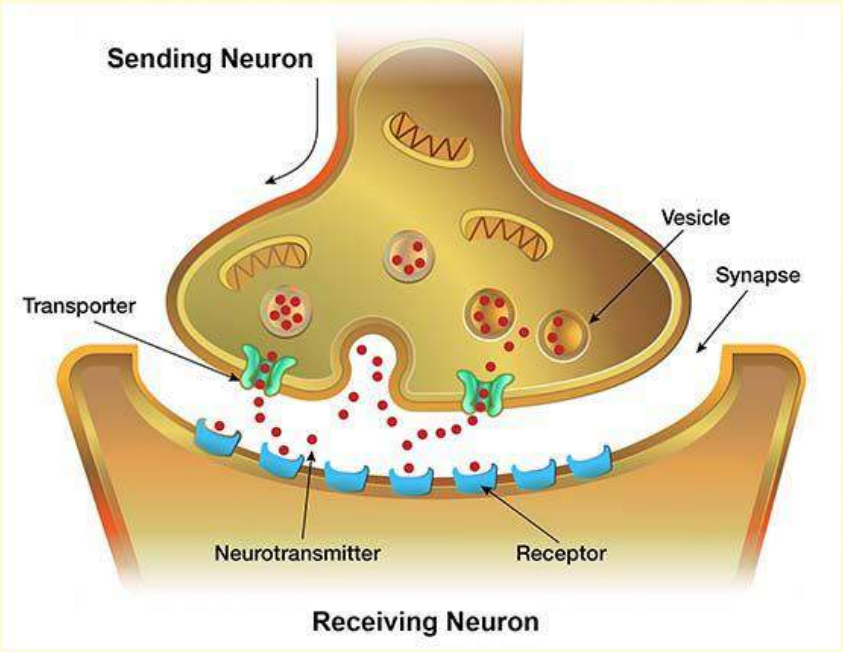
- junction between neurons
- has to jump quickly from one cell to next

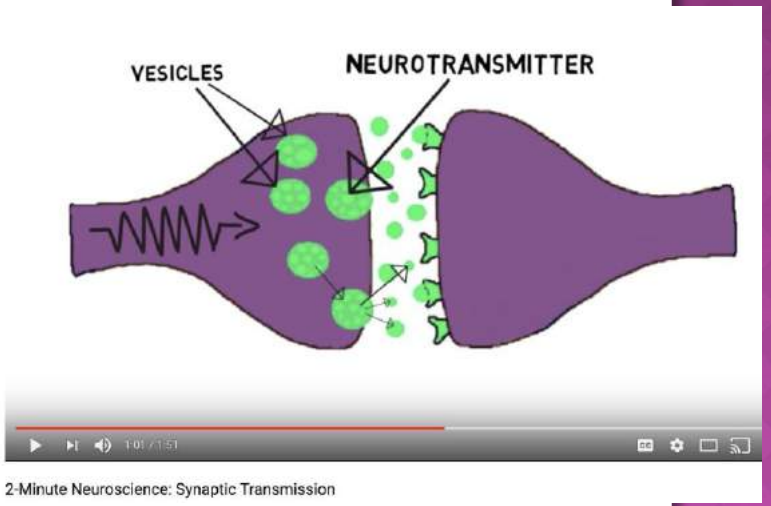
SYNAPSE

Synaptic Transmission

At the dendritic end of the nerve cell. Each dendrite collects the nerve impulse From the terminal end of a different nerve cell. The impulse needs to jump across a small gap called the synapse… by a synaptic transmission.

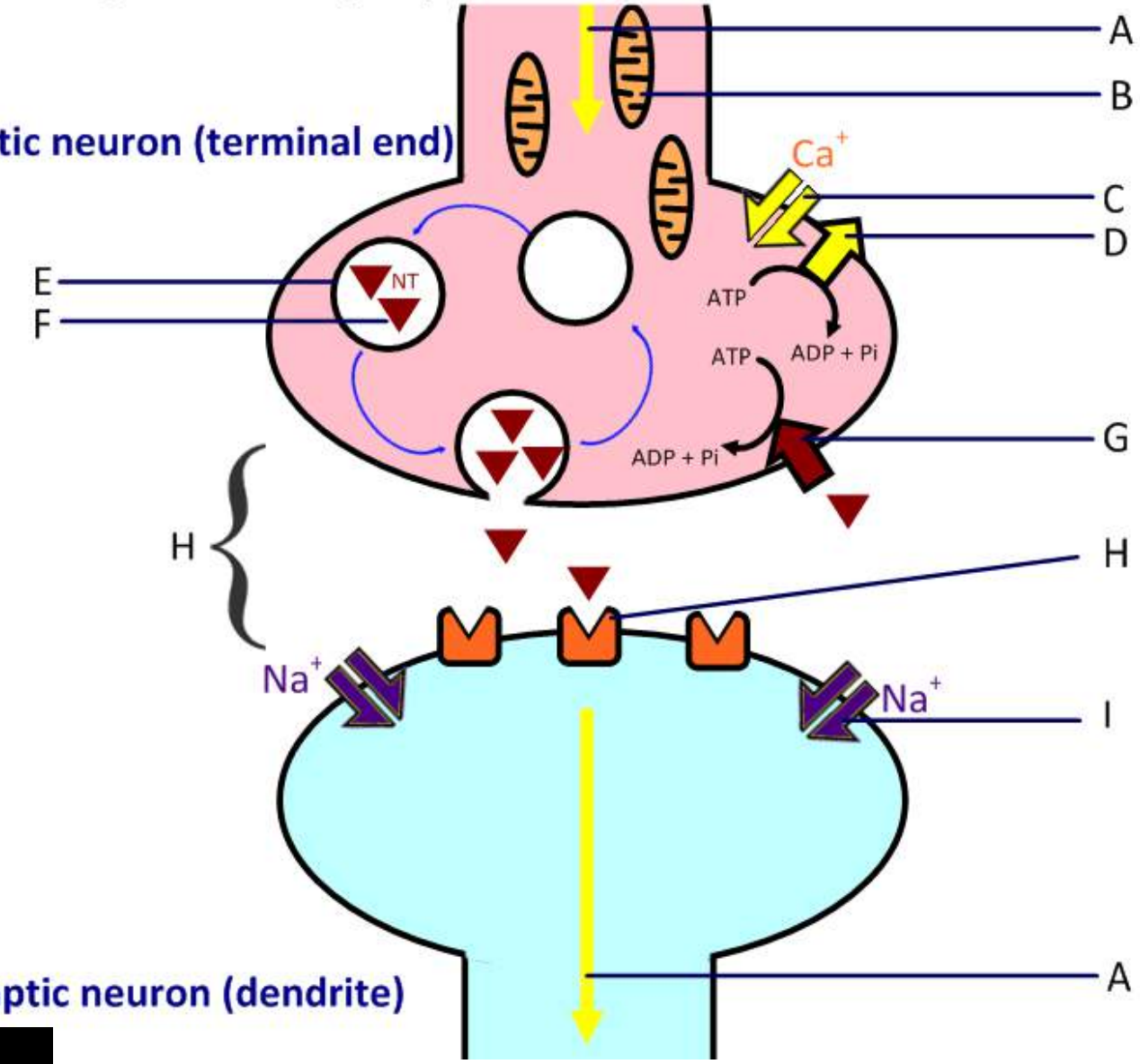
Its an electrical impulse converted to a chemical neurotransmitter.





2-Minute Neuroscience: Synaptic Transmission

Label this diagram of a synapse



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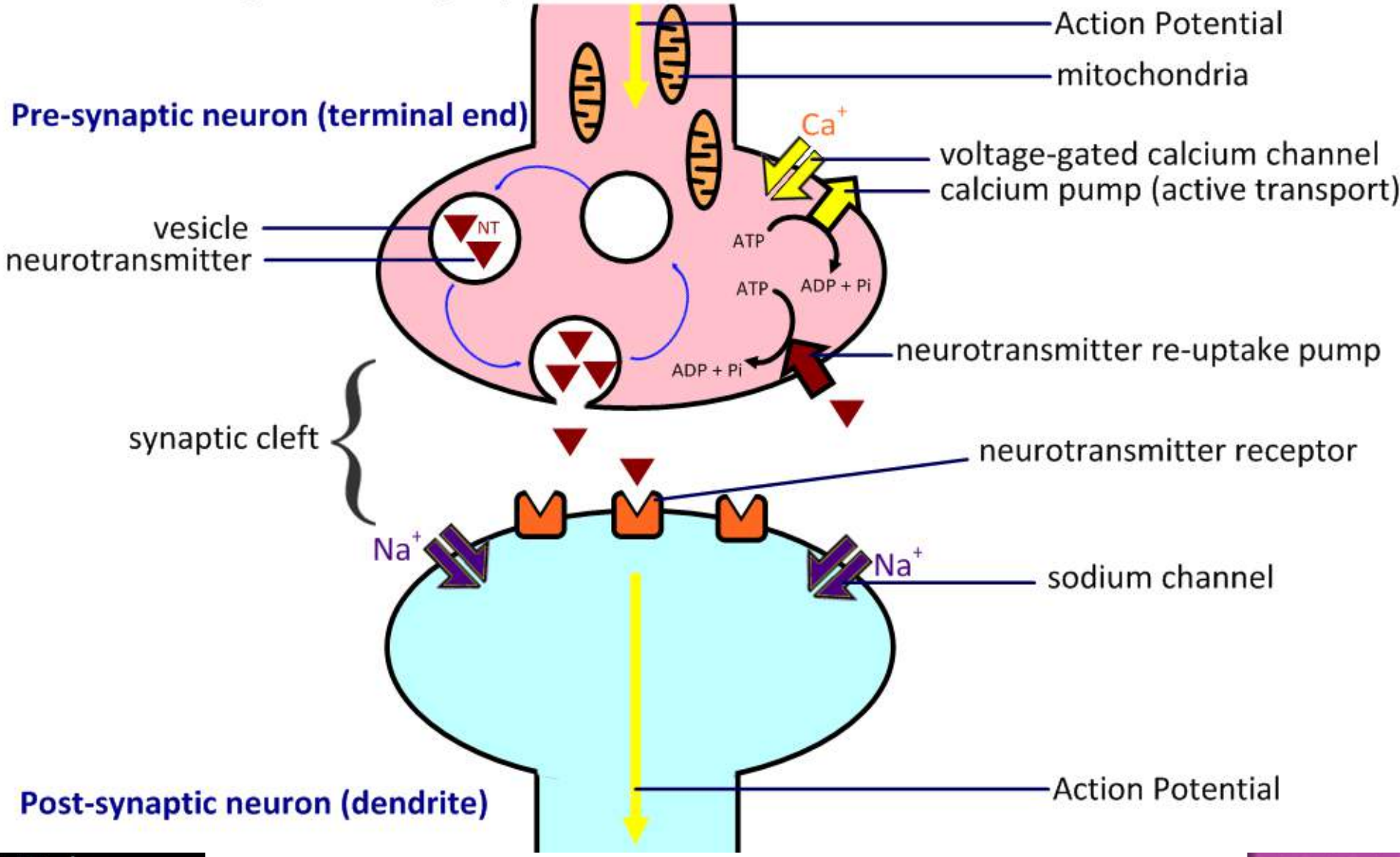
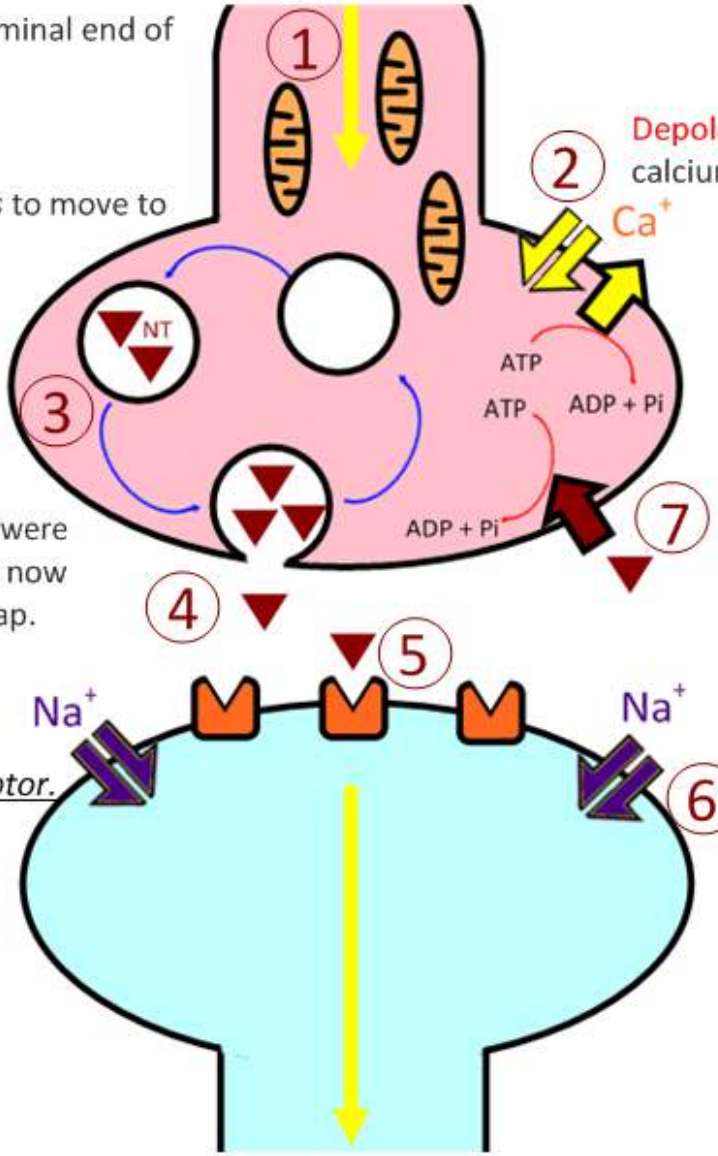


Summary of Synaptic Transmission

1. Nerve impulse reaches terminal end of pre-synaptic neuron.

2. Depolarisation causes voltage-gated calcium channels to open. Ca^{2+} rushes in.

3. Ca^{2+} causes *synaptic vesicles* to move to the membrane and fuse.



4. Neurotransmitters (NTs)that were stored in the synaptic vesicle now diffuse across the synaptic gap.

5. NTs bind with post-synaptic receptors.
NTs are specific to the receptor.

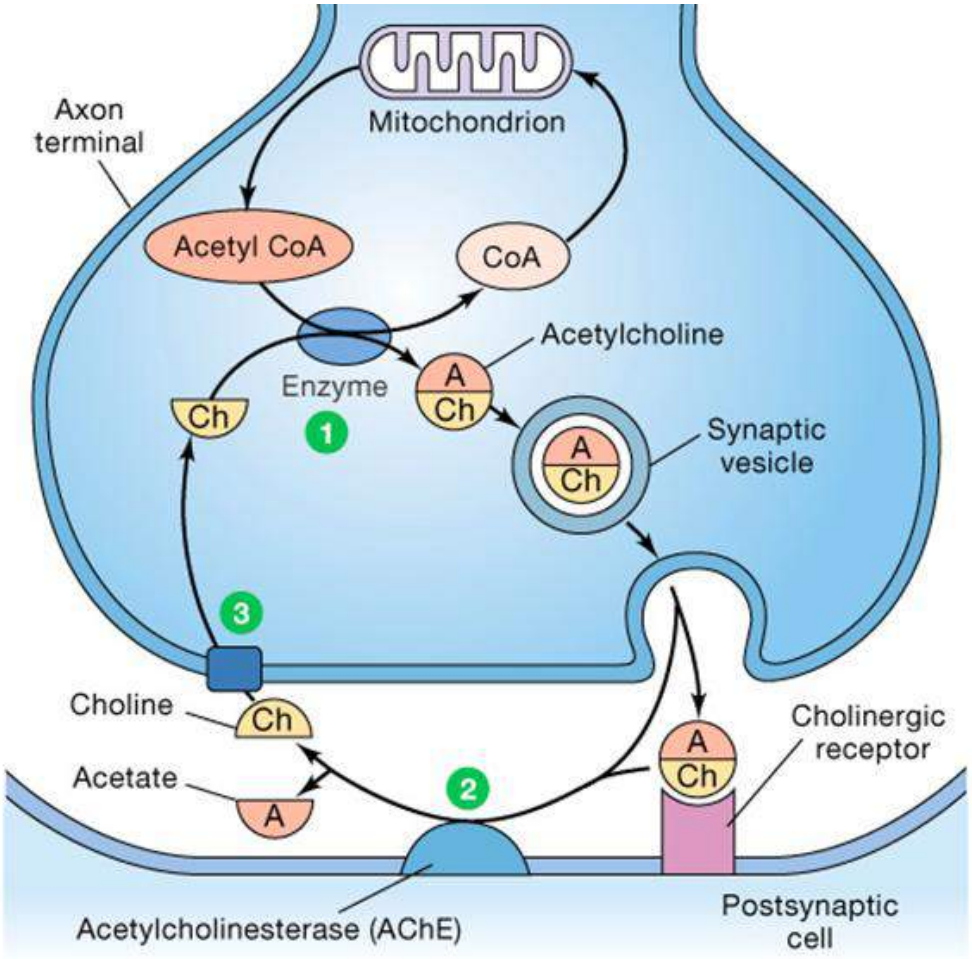
6. Sodium channels open, causing Na^{+} to enter, leading to depolarisation of the post-synaptic neuron.
An action potential is initiated.

7. Enzymes in the synaptic gap then break down the NT.
The products of this break down are taken up by the pre-synaptic neuron by active transport (hence the large number of mitochondria)

The nerve impulse is then propagated along the post-synaptic neuron.

Acetylcholine is a neurotransmitter used in many synapses through the nervous system

Cholinergic synapses



1. **Acetylcholine** (ACh) is made from choline and acetyl CoA.
2. In the synaptic cleft ACh is rapidly broken down by the enzyme **acetylcholinesterase.**
3. Choline is transported back into the axon terminal and is used to make more ACh.

One use is at the neuromuscular junction, i.e. it is the molecule that motor neurons release to activate muscles. Interfering with the action of acetylcholine can cause a range of effect from paralysis to convulsions.

What are Neonicotinoids?

- Pesticides that effect a wide range of crop pests.
- They are the most widely used insecticides in the world accounting for 25% of all insecticides used.

How Do Neonicotinoids Work?

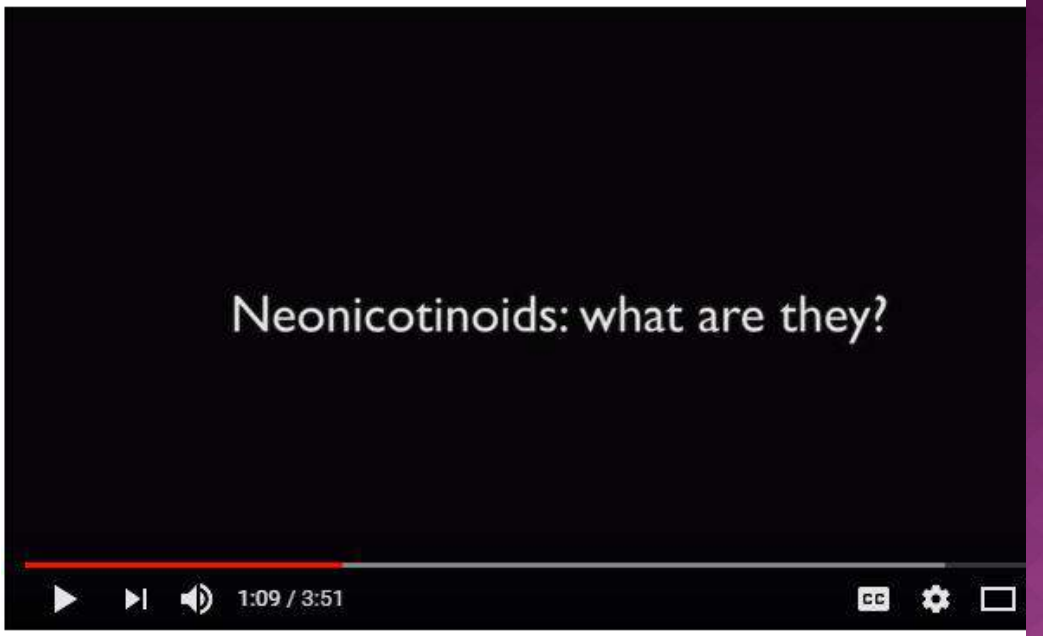
- This pesticide can be added to the water during irrigation. This provides better plant tissue contact.
- An insecticide that can be applied to seeds before planting.
- Neonicotinoids bind to the insects receptors for their neurotransmitters. This overstimulates and blocks receptors leading to death

Blocking of synaptic transmission at cholinergic synapses in insects by binding of neonicotinoid pesticides to acetylcholine receptors.

Environmental Concerns for Neonicotinoids

- Neonicotinoids can accumulate in the soil.
- Increasing evidence the pesticide is affecting non-target Organisms.

Examples are the honey bee and monarch butterfly populations



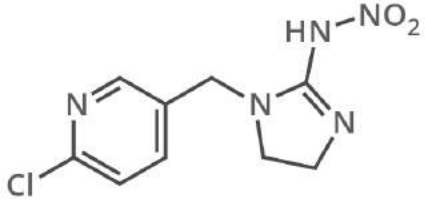
Quantifying the effects of Neonicotinoids on honeybees and wild bees: a Europe-wide experiment

The Death Of Bees Explained – Parasites, Poison and Humans

Blocking of synaptic transmission at cholinergic synapses in insects by binding of neonicotinoid pesticides to acetylcholine receptors.

NEONICOTINOID PESTICIDES - THE FACTS

The use of neonicotinoid pesticides has been a contentious issue in recent years. They account for around 25% of the global agrochemical market, but have also been linked with negative environmental effects. This graphic looks at how they work, and the nature of the concerns surrounding them.



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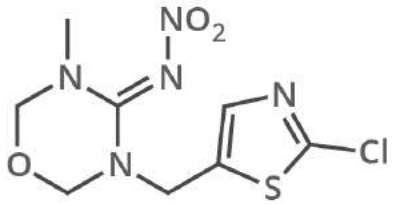

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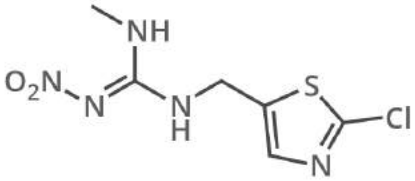


CLOTHIANIDIN

1980s Decade in which neonicotinoid pesticides first developed

120 Number of countries in which neonicotinoids are registered

Now used more than any other class of insecticide.

HOW DO NEONICOTINOIDS WORK?

Can be added to irrigation water, then taken up & spread through plant tissues. Also used in seed treatments.

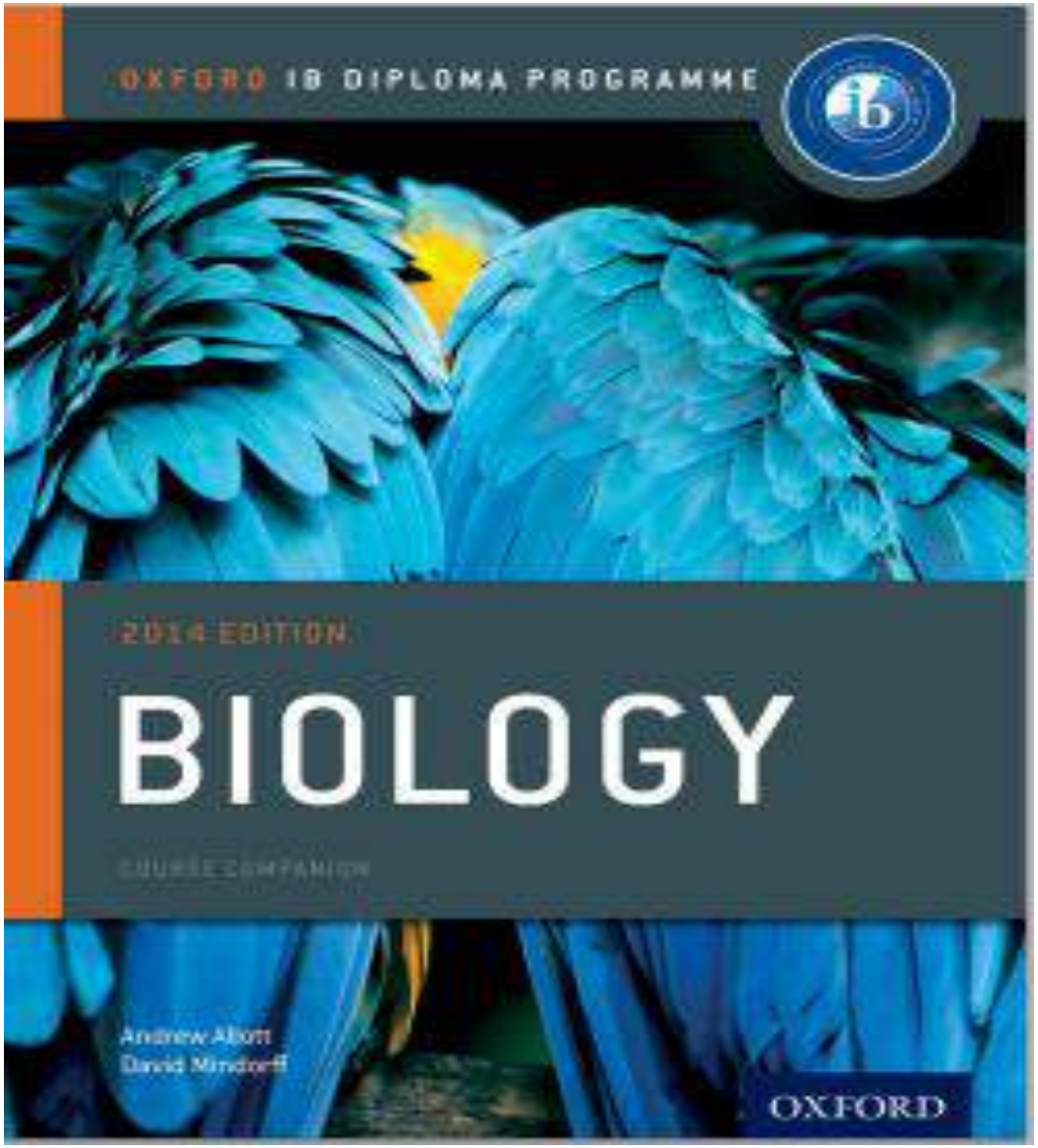
Bind to nicotinic receptors for the neurotransmitter acetylcholine in the insect central nervous system.

This leads to overstimulation and blocking of the receptors, leading to paralysis and eventual death.

Neonicotinoids pesticides are effective against a wide range of crop pests. They are the most widely used insecticides in the world, accounting for roughly 25% of all insecticide use. Median lethal doses vary depending on the size of the insect, ranging from less than 1 nanogram to almost 90 nanograms per insect. Mammals also have the receptors neonicotinoids bind to, but they bind to them less strongly than in insects, so neonicotinoid mammalian toxicity is much lower.

ENVIRONMENTAL CONCERNS

- Can accumulate in soil; low concentrations found in nectar of treated crops.
- Linked as contributors to honey bee colony decline. However, this is still inconclusive, and subject to continued research and conflicting interpretations.
- Increasing evidence of effects on non-target organisms. Negative impacts on monarch butterfly populations in the USA have recently been suggested.
- Use has been partially restricted in the EU since 2013. However, some have suggested this has merely led to increased use of older, harsher pesticides.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