

- Draw a basic neuron. What are the different parts, and what, very generally speaking, do they do?
  - Dendrite: receive inputs
  - Cell body: integrate inputs
  - Axon: conduct a signal
  - Synapse: output the signal
- Do all neurons connect to other neurons?
  - No! Neurons can have their output onto muscle cells, endocrine cells, etc.
- What is the electrical state of a neuron at rest? How is this state maintained?
  - It is polarized: more negative inside the neuron than outside the neuron.
  - This is because of:
    - Leak channels
    - The sodium potassium ATPase
    - The presence of large, negatively charged proteins inside the neuron
- What does the sodium potassium ATPase do? How does it work?
  - It uses ATP to pump three sodium ions out of the cell and pump two potassium ions into the neuron, creating the asymmetric distribution of these ions on either side of the cell membrane
- Why are neurons energetically demanding cells?
  - Because they must do a lot of active transport of ions against their electrochemical gradient.
- The balance between an \_\_\_\_\_ gradient and a \_\_\_\_\_ gradient produces the equilibrium potential of a charged ion across a permeable membrane.
  - This is the balance between the electrical gradient, which wants overall electrical neutrality (because like charges repel and opposite charges attract), and the chemical gradient, which wants equal concentration of the ion on both sides of the membrane
- What equation can be used to find the equilibrium potential for a single ion? Define each term, and explain two parameters that go into determining the constant for an ion.
  - The equilibrium potential can be found from the Nernst equation:  $E_{ion} = \text{constant} \times \log(\text{concentration outside}/\text{concentration inside})$
  - Constant is determined by:
    - The temperature
    - The charge of the particular ion in question
- What are the two types of membrane potential? Cells can produce a \_\_\_\_\_ potential or an \_\_\_\_\_ potential.
  - Graded and action potentials
- What is a graded hyper- or depolarization? What channels cause them? What stimuli would they occur in response to? What can be the result of them (discuss spatial/temporal summation here).
  - Graded potentials are smaller changes in membrane potential that can vary in their magnitude

- They can be depolarizations, which get the neuron closer to its threshold potential and are therefore excitatory, or hyperpolarizations, which take the neuron farther away from the threshold potential and are therefore inhibitory.
- They are caused by ions flowing through ligand-gated channels
- They occur in response to neurotransmitter release from a neighboring neuron
- If the sum of all the graded potentials in a neuron reaches the threshold potential, then an action potential will be generated; otherwise the neuron will not fire.
- Graded hyperpolarizations
  - Produced by stimuli that increase the membrane's permeability to potassium ( $K^+$ )
  - Results from ion movement through ligand gated channels
- Graded depolarization
  - Produced by stimuli that increase the membrane's permeability to sodium ( $Na^+$ )
  - Results from ion movement through ligand gated channels
- What must happen for a neuron to be triggered to fire an action potential?
  - It must reach the threshold voltage, the specific membrane voltage that triggers opening of voltage gated channels
- Describe the phases that a neuron goes through as it moves from rest through firing an action potential back to rest. Explain how channels open and close, which ions flow in what direction when, and how the membrane voltages changes as a result.
  - Steps:
    - Resting potential: voltage gated sodium and potassium channels are closed
    - Depolarization to threshold: some ligand-gated sodium channels open and let sodium into the cell. This decreases the polarization of the membrane from -70 mV to -50 mV, the threshold potential
    - Action potential:
      - Depolarization, aka rising phase: once the threshold potential is reached, that voltage triggers all of the voltage-gated sodium channels to open all at once. This allows the membrane potential to shoot up to +40 mV.
      - Repolarization, aka falling phase:
        - The same voltage level triggers both sodium channels and potassium channels to open. However, sodium channels open a lot faster than potassium channels; that is what creates the rising phase and then the falling phase of the action potential.
    - Hyperpolarization, aka overshoot:
- What properties of the neuron and the action potential ensure that action potentials move in only one direction down the axon (ie don't move backward)?

- Diffusion of sodium inside of the neuron helps the next patch of the neuron reach its threshold potential, triggering an action potential at the next part of the neuron
- The action potential only moves in one direction because the overshoot/hyperpolarization prevents sodium diffusion from causing the cell to reach the threshold potential when it has just experienced an action potential
- What is the effect of axon diameter on rate of action potential propagation? What kinds of behaviors would be controlled by neurons with larger diameters?
  - The bigger the neuron, the faster the action potential can travel down the neuron
  - So you would use big neurons to control behaviors that need to happen really quickly, like an escape-from-predator reflex
- Can an action potential generated by one patch of the neuronal membrane travel infinitely down the axon?
  - No. Action potentials decay, and must be re-propagated by more voltage-gated channels in order to make it all the way down the neuron.
- What controls the rate of action potential decay (two factors)?
  - Axon diameter (squared)
  - Resistance across the membrane, which is a function of what ion channels in the membrane are open
- How do neurons prevent action potentials from decaying to zero and make sure that the action potential is propagated all along the axon? Think about the two factors that control rate of decay, and how cells can manipulate these two factors.
  - Rate of decay depends on axon diameter and membrane resistance.
  - Can make the diameter bigger
  - Membrane resistance:
    - Glial cells wrap internodes of the axon in layers and layers of myelin, basically layers of phospholipid bilayer, leaving nodes exposed. Those nodes are where the voltage gated channels are found, and the action potential is re-propagated.
      - Myelin decreases resistance of the membrane by preventing any channel activity, allowing the signal to move faster
    - This allows saltatory conduction- jumping conduction- where the signal jumps through the internodes and is re-propagated as an action potential by voltage gated channels in the node
- What happens if myelin breaks down?
  - Axons break down
- How does lidocaine work to prevent you from feeling pain?
  - It blocks voltage-gated sodium channels in pain nerves, so that those nerves cannot fire a signal of pain to the brain
- How does scorpion venom cause pain? How do grasshopper mice resist this venom?
  - Bark scorpion venom binds to voltage-gated sodium channels in pain nerves, opening them and making the nerve fire excessively, causing tremendous pain
  - Grasshopper mice have a mutation in their voltage-gated sodium channels so that the venom does not cause them to open excessively