
Figure 35.4
Biology: How Life Works
© 2014 W. H. Freeman and Company
Squid Giant Axon

The Na⁺-K⁺ pump moves Na⁺ ions out of the cell and K⁺ ions into the cell.

K⁺ channels allow K⁺ ions to "leak" out of the cell, resulting in a negative resting potential on the inside relative to the outside of the cell.

Membrane potential can be measured with small glass electrodes.

Figure 35.8
Biology: How Life Works
© 2014 W. W. Freeman and Company
Diagram 35.9

1. Summed input depolarizes the cell membrane at the axon hillock above the threshold potential.

2. Voltage-gated Na⁺ channels open and Na⁺ enters the cell, causing a positive spike in the membrane potential (positive inside relative to outside).

3. As the voltage rises to +40mV, Na⁺ channels close and voltage-gated K⁺ channels open, allowing K⁺ ions to leave the cell and causing the membrane potential to become more negative.

4. An overshoot in the amount of K⁺ ions that leave the cell causes the cell membrane to be hyperpolarized. This results in a refractory period during which the nerve cannot fire another action potential.

5. Gradually the membrane returns to resting, as excess K⁺ ions are returned to the cell.

Diagram 35.10a

Extracellular fluid

Voltage-gated K⁺ channel

Voltage-gated Na⁺ channel

Membrane

Cytoplasm

Depolarizing

Resting
Figure 35.10b
Biology: How Life Works
© 2014 W. H. Freeman and Company

Repolarizing

Depolarizing

Resting

Figure 35.11
Biology: How Life Works
© 2014 W. H. Freeman and Company

Layers of myelin insulate the axon. As a result, action potentials “jump” from node to node, increasing the speed of conduction.

At nodes of Ranvier, there is a buildup of + charges inside and – charges outside the axon.
MRI/PET combo


Synapse Types

1. Synaptic transmission begins with action potential conduction to the axon terminal.

2. Depolarization of the axon terminal opens voltage-gated Ca²⁺ channels.

3. Vesicles respond by fusing with the presynaptic membrane, releasing neurotransmitters into the synaptic cleft.

4. Neurotransmitters bind with receptors on the postsynaptic cell, causing a change in membrane potential.

5. After inactivation, neurotransmitter molecules are re-absorbed into the presynaptic terminal and stored in vesicles until the next action potential arrives.

---

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.
Each yellow-green dot represents an axon terminal from another neuron synapsing on the dendrites of this (green) postsynaptic cell.
Figure 36.3
Biology: How Life Works
© 2014 W. H. Freeman and Company

Action potential firing rates correlate with the intensity of the stimulus.

Adaptation to continuous stimuli reduces the firing rate over time.

Figure 36.4
Biology: How Life Works
© 2014 W. H. Freeman and Company

Lateral inhibition of sensory receptor cells enhances edge and border detection by reducing excitatory stimulus of adjacent interneurons.
Peptide neuromodulators

Figure 36.25
Biology: How Life Works
© 2014 W. H. Freeman and Company

Repeated release of the neurotransmitter glutamate stimulates the production of new glutamate receptors, increasing the strength of the signals received by the cell.

Repeated stimulation also induces new dendritic synapses to grow, further strengthening the signal connection between the two cells and thereby reinforcing a particular memory.

Alternative mRNA splicing of the FMRFamide gene

A. Primary transcript

- Exon I
- FMRFa
- Intron
- Exon II

B. Alternative splicing

- mRNA 1
  - Exon I
  - II
- mRNA 2
  - Exon I
  - II

C. Translation

- Protein precursor 1
  - Leader peptide
  - FLRFa
  - EFLRfa
  - SDPYLRFa
  - SDPFLR Fa
  - SPYFMRFa
  - Acidic peptide (P3 + P1)
  - SDPFFRGKQVATDSDGELDELRSRVSDDDKNI
  - pQFYRla
  - SDPYLRFa
  - SEQPDVEDLKDVLQISELEY
  - pQQVATDSDLDELRSVSDDDKNI (P1)
- Protein precursor 2
  - 22 aa peptide (P2)

DOI: 10.4249/scholarpedia.11520