

Intracellular Recording

**Application:** Electrophysiologists use intracellular recording to measure the membrane potential of neurons and other cells.

**Technique:** A microelectrode is made from a glass capillary tube filled with an electrically conductive salt solution. One end of the tube tapers to an extremely fine tip (diameter < 1 μm). While looking through a microscope, the experimenter uses a micropositioner to insert the tip of the microelectrode into a cell. A voltage recorder (usually an oscilloscope or a computer-based system) measures the voltage between the microelectrode tip inside the cell and a reference electrode placed in the solution outside the cell.



to that of  $K^+$  (see Table 48.1).  $Na^+$  therefore diffuses into the cell, making the inside of the cell less negative. If we model a membrane in which the only open channels are selectively permeable to  $Na^+$ , we find that a tenfold higher concentration of  $Na^+$  in the outer chamber results in an equilibrium potential ( $E_{Na}$ ) of +62 mV (Figure 48.7b). In an actual neuron, the resting potential (−60 to −80 mV) is much closer to  $E_K$  than to  $E_{Na}$  because there are many open potassium channels but only a small number of open sodium channels.

Because neither  $K^+$  nor  $Na^+$  is at equilibrium in a resting neuron, there is a net flow of each ion (a current) across the membrane. The resting potential remains steady, which means that the  $K^+$  and  $Na^+$  currents are equal and opposite. Ion concentrations on either side of the membrane also remain steady. Why? The resting potential arises from the net movement of far fewer ions than would be required to alter the concentration gradients.

Under conditions that allow  $Na^+$  to cross the membrane more readily, the membrane potential will move toward  $E_{Na}$  and away from  $E_K$ . As you'll see in the next section, this is precisely what happens during the generation of a nerve impulse.

CONCEPT CHECK 48.2

- Under what circumstances could ions flow through an ion channel from a region of lower ion concentration to a region of higher ion concentration?
- WHAT IF?** Suppose a cell's membrane potential shifts from −70 mV to −50 mV. What changes in the cell's permeability to  $K^+$  or  $Na^+$  could cause such a shift?
- MAKE CONNECTIONS** Review Figure 7.10, which illustrates the diffusion of dye molecules across a membrane. Could diffusion eliminate the concentration gradient of a dye that has a net charge? Explain.

For suggested answers, see Appendix A.

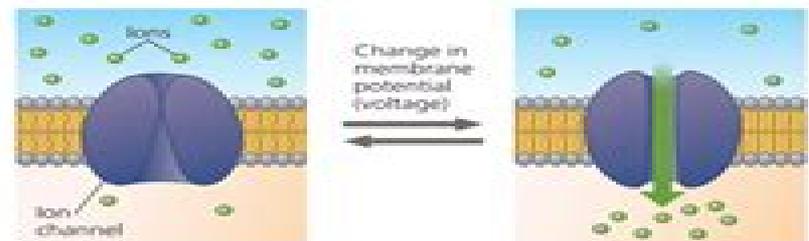
CONCEPT 48.3

Action potentials are the signals conducted by axons

When a neuron responds to a stimulus, such as the scent of fish detected by a hunting cone snail, the membrane potential changes. Using the technique of intracellular recording, researchers can record these changes as a function of time (Figure 48.8). Changes in the membrane potential occur because neurons contain **gated ion channels**, ion channels that open or close in response to stimuli. The opening or closing of gated ion channels alters the membrane's permeability to particular ions, which in turn alters the membrane potential.

Hyperpolarization and Depolarization

When gated ion channels are stimulated to open, ions flow across the membrane, changing the membrane potential (Figure 48.9). For example, opening gated potassium channels in a resting neuron increases the membrane's permeability to  $K^+$ . Net diffusion of  $K^+$  out of the neuron increases, shifting the membrane potential toward  $E_K$  (−90 mV at 37°C). This increase in the magnitude of the membrane potential, called a **hyperpolarization**, makes the inside of the membrane more negative (Figure 48.10a). In a resting neuron, hyperpolarization results from any stimulus that increases the outflow of positive ions or the inflow of negative ions.



Gate closed: No ions flow across membrane.

Gate open: Ions flow through channel.

**▲ Figure 48.9 Voltage-gated ion channel.** A change in the membrane potential in one direction (indicated by the right-pointing arrow) opens the voltage-gated channel. The opposite change (left-pointing arrow) closes the channel.

# 48 Neurons Synapses And Signaling Guide Answers

**Jacquelyn L. Banasik**



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Spike-timing dependent plasticity Henry Markram, Wulfram Gerstner, Per Jesper Sjöström, Hebb's postulate provided a crucial framework to understand synaptic alterations underlying learning and memory Hebb's theory

proposed that neurons that fire together also wire together which provided the logical framework for the strengthening of synapses Weakening of synapses was however addressed by not being strengthened and it was only later that the active decrease of synaptic strength was introduced through the discovery of long term depression caused by low frequency stimulation of the presynaptic neuron In 1994 it was found that the precise relative timing of pre and postsynaptic spikes determined not only the magnitude but also the direction of synaptic alterations when two neurons are active together Neurons that fire together may therefore not necessarily wire together if the precise timing of the spikes involved are not tightly correlated In the subsequent 15 years Spike Timing Dependent Plasticity STDP has been found in multiple brain regions and in many different species The size and shape of the time windows in which positive and negative changes can be made vary for different brain regions but the core principle of spike timing dependent changes remain A large number of theoretical studies have also been conducted during this period that explore the computational function of this driving principle and STDP algorithms have become the main learning algorithm when modeling neural networks This Research Topic will bring together all the key experimental and theoretical research on STDP

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**Discover**, 1989

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determine the structure and function of an organ such as the brain. It is therefore an essential process linking molecular genetics with neurochemistry and behavioral neuroscience. This volume presents a didactic approach to the understanding of the basic processes of gene expression and their involvement in certain brain diseases such as Alzheimer's disease and schizophrenia. Generously illustrated, the contributions provide a valuable outline of this key aspect of molecular neurobiology and clinical neuroscience.

*Review of Medical Physiology* Edward G. Schneider, Steven L. Bealer, 1998. This review and study guide is designed for medical students who are using the Second Edition of Johnson's *Essential Medical Physiology* in their course work. The review book contains 1,000 questions based on the text plus answers and discussions. Also included are 62 illustrative drawings, most of them selected from the text. Other user-friendly features include detailed outlines of the chapters of the text and boxed displays containing hot topics or key facts/statements. Used in conjunction with *Essential Medical Physiology*, this review book will stimulate deeper engagement with the text, serve as a guide to key points, and refresh the student's memory before an exam.

**Fundamental Neuroscience** Michael J. Zigmond, 1999. *Fundamental Neuroscience* is a comprehensive textbook that seeks to define the full scope of neuroscience. Developed in accordance with results of extensive reviews by neuroscience instructors, this premier textbook is divided into seven integrated sections. Each section may be used for a specific course or the full text may be adopted to provide a broad-based curriculum that will carry the student from molecular to cognitive neuroscience.

**Psychopharmacology Bulletin**, 1966. **Science** John Michels (Journalist), 2001. *Scientific American*, 1987. Monthly magazine devoted to topics of general scientific interest.

**Neurobiological Basis of Learning and Memory** Yasuzō Tsukada, Bernard W. Agranoff, 1980. Unlisted Drugs, 1949. Investigative Ophthalmology & Visual Science, 2000.

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