Synapse

This article describes in detail the chemical synapses and not electrical synapses. Chemical synapses are the ones found between axon terminals and dendrites of neurons (pre/post synaptic membranes are not in contact), whereas electrical synapses are the ones where the pre- and post-synaptic membranes are in contact and the current is passed through gap junctions (such as in cardiomyocytes).

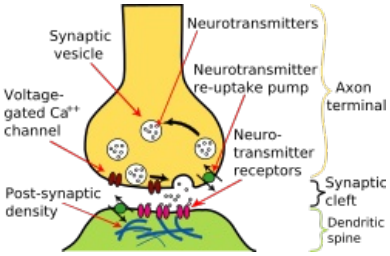
Electrical Synapses

The simplest way for one neuron to pass its signal to another is by direct electrical coupling through low resistance pathways of gap junctions. Such electrical synapses transmit without delay and in both directions, but the integration properties of such synapses are very limited. In nonneuronal elements the gap junction coupling is present among myocardial cells, intestinal smooth muscle cells, hepatocytes, etc.

Chemical Synapses

Structure

Chemical synapses, in contrast to electrical synapses, provide a wider spectrum of possibilities for adjustment and control of the signal transmission. The principles of chemical communication at a synapse are the same as those of chemical communication by water-soluble hormones. The proximity between the 2 ends of the synapse is main reason why the neurotransmitters released are much less diluted leading to a very effective transmission. Due to the inherent organization of the chemical synapse, conduction is one way, but with a certain delay.



Chemical Synapse Overview

When a neuron makes a chemical synapse with another neuron, the presynaptic nerve terminal characteristically broadens to form a terminal bouton. The number of these terminals range from 10000 to 200000 laying over the dendrites (80-85%) and the rest over the soma (5-20%). Areas of high electron density adjacent to the plasma membranes in the region of synaptic contact are visible in EM (symmetrical and asymmetrical synapses, Gray I and Gray II synapses).

Function

Membrane potential changes of the presynaptic terminal (e.g., action potential) cause the release of a neurotransmitter by exocytosis, due to the activation of Ca^{2+}-VGCs. The inflowing calcium ions bind on special protein molecules on the inside surface of the membrane, called released sites, which causes them to open and release the vesicles. Each vesicle can release from 2000 to 10000 molecules of neurotransmitter. After the release, each of them is recycled and refilled with new neurotransmitter substance. For the synthesis of the neurotransmitter and its packaging into vesicles, ATP is used (which is provided by the numerous mitochondria in the vicinity of the presynaptic membrane).

The neurotransmitter diffuses across the synaptic cleft (about 50 nm) and binds to a specific membrane protein (receptor). Post-synaptic membranes have receptor proteins. These proteins have:

- Binding component, which will accept the neurotransmitter on the exterior surface and this will cause a conformational change on the ionophore component.
- Ionophore component, which passes through the post-synaptic membrane and into the interior of the neuron. It can be one of two types:
    1. Ion channel (2 types): they open/close in less than a ms, upon presence/absence of transmitter
        1. Cation channels: conduct Na^+ into the cell. They are lined with negative charges thus attracting the positive sodium ions. When the diameter of the channel is greater or equal to the diameter of the hydrated sodium ion, conduction occurs. The negative charges at the same time repel anions, such as Cl^-. These channels contribute to excitatory Post-Synaptic-Potentials (EPSPs), by inducing depolarization on the intracellular fluid of the dendrite/soma.
        2. Anion channels: They allow chloride ions to pass though, while blocking passage of cations (Na, K, Ca), mainly because their hydrated forms are bigger than the channel's diameter. These channels contribute to inhibitory Post-Synaptic Potentials (IPSPs), by inducing hyperpolarization on the intracellular fluid of the dendrite/soma.
    2. Second Messenger activator: (slow response with long-lasting duration): G-proteins, upon binding with the neurotransmitter, release their α-subunit into the cytoplasm in the interior of the post-synaptic membrane. The α-subunit can perform multiple actions to the cell's structure/metabolism, thus causing a prolonged effect to the cell that might range from seconds to months. Possible actions are:
        1. Opening specific ion channels (e.g.: Ca2+ or K+) through the postsynaptic cell membrane. This ion channel often stays open for a long time, thus causing long-term changes to the electric potential inside the cell.
        2. Activation of cAMP or cGMP or Inositol triphosphate. These molecules can in turn activate highly specific metabolic machinery → initiate chemical reactions that can affect the cell's structure →

affecting the long-term excitability of the neuron.
3. Activation of one or more intracellular enzymes. These enzymes can in turn catalyze various chemical reactions which in the end can affect the neuron's future excitability.
4. Activation of gene transcription. This can lead to formation of new receptors that might cause easier inhibition or easier excitation (facilitation) of the neuron. This is an important mechanism not only for memory but also for up-regulation and down-regulation of specific receptors, according to the frequency the neuron is excited or inhibited.

The termination, in other words the removal of the neurotransmitter from the receptor, can occur by at least one of the following ways:

1. By breaking away from the receptor due to thermally-induced oscillations that originate from the neurotransmitter itself and the receptor. This will allow the enzymes (such as acetylcholinesterase) to breakdown the neurotransmitter in the synaptic cleft or be up-taken in the presynaptic membrane by a reuptake pump.
2. Various enzymes present within the subsynaptic membrane may inactivate/metabolize the neurotransmitter.

Links

Related articles

- Synaptic Transmission

Sources

- Lecture Notes: Prof. MUDr. Jaroslav Pokorný DrSc.

Bibliography

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Further reading