Synaptic transmission: A comprehensive review.

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Introduction

Synaptic transmission, the process by which neurons communicate with each other, lies at the heart of nervous system function. This review aims to provide a comprehensive overview of the intricate mechanisms underlying synaptic transmission, from the release of neurotransmitters to the modulation of synaptic strength. Understanding synaptic transmission is essential for unravelling the complexities of brain function and for developing treatments for neurological disorders [1, 2].

At the synaptic terminal, neurotransmitter release occurs in a highly orchestrated manner. Action potentials trigger the opening of voltage-gated calcium channels, leading to calcium influx into the presynaptic terminal. This influx triggers the fusion of neurotransmitter-containing vesicles with the presynaptic membrane, releasing neurotransmitters into the synaptic cleft. The release process is tightly regulated by various proteins and signalling molecules, ensuring precise control over synaptic communication [3].

Upon release, neurotransmitters bind to specific receptors on the postsynaptic membrane, initiating a cascade of intracellular signalling events. These receptors can be classified into two main types: ionotropic receptors, which directly gate ion channels, and metabotropic receptors, which activate intracellular signalling pathways *via* G proteins. The diversity of neurotransmitter receptors allows for finetuning of synaptic transmission and enables complex neuronal signalling [4].

Synaptic transmission is not static but subject to modification in response to activity patterns and environmental stimuli. Synaptic plasticity, the ability of synapses to change their strength, underlies learning and memory processes in the brain. Long-Term Potentiation (LTP) and Long-Term Depression (LTD) represent two well-characterized forms of synaptic plasticity, wherein sustained changes in synaptic strength occur through alterations in receptor trafficking, synaptic morphology, and neurotransmitter release probability [5].

Synaptic transmission is subject to modulation by a variety of factors, including neuromodulators, hormones, and drugs. Neuromodulators such as dopamine, serotonin, and acetylcholine can alter synaptic strength and neuronal excitability, thereby influencing behavior and cognition. Additionally, drugs targeting specific neurotransmitter systems, such as antidepressants and antipsychotics, exert their therapeutic effects by modulating synaptic transmission [6, 7].

Dysregulation of synaptic transmission underlies numerous neurological disorders, including epilepsy, Parkinson's disease, and Alzheimer's disease. Understanding the mechanisms of synaptic dysfunction in these disorders is critical for developing targeted therapeutic interventions aimed at restoring normal synaptic function. Emerging treatments targeting synaptic transmission hold promise for improving the lives of individuals affected by neurological disorders [8, 9].

Synaptic transmission represents a fundamental process in nervous system function, enabling communication between neurons and the integration of neural circuits. By elucidating the mechanisms underlying synaptic transmission and its modulation, researchers can gain insights into brain function and dysfunction. Continued research in this field promises to uncover novel therapeutic strategies for treating neurological disorders and enhancing cognitive function [10].

References

- 1. Marosi EL, Arszovszki A, Brunner J, et al. Similar presynaptic action potential-calcium influx coupling in two types of large mossy fiber terminals innervating CA3 pyramidal cells and hilar mossy cells. Eneuro. 2023;10(2).
- 2. Biswas P, Karan P, Pal S, et al. Smartphone-Interfaced Serum Calcium Level Quantification on a Simple Paper Strip Assay for Diagnostics at Extreme Point of Care. IEEE J-FLEX. 2023.
- 3. Ruach R, Yellinek S, Itskovits E, et al. A negative feedback loop in the GPCR pathway underlies efficient coding of external stimuli. Mol Syst Biol. 2022;18(9):e10514.
- 4. Zhang Y, Li D, Darwish Y, et al. KCNQ channels enable reliable presynaptic spiking and synaptic transmission at high frequency. J Neurosci Res. 2022;42(16):3305-15..
- 5. Ormerod KG, Scibelli AE, Littleton JT. Regulation of excitation-contraction coupling at the Drosophila neuromuscular junction. Physiol J. 2022;600(2):349-72.
- 6. Kang Y, Jang J, Cha D, et al. Synaptic weight evolution and charge trapping mechanisms in a synaptic passtransistor operation with a direct potential output. IEEE Trans Neural Netw Learn Syst. 2021;32(10):4728-41.

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- 7. Shvarts-Serebro I, Sheinin A, Gottfried I, et al. miR-128 as a Regulator of Synaptic Properties in 5xFAD Mice Hippocampal Neurons. J Mol Neurosci. 2021;71:2593-607.
- Velásquez A, Chamorro Y, Maldonado A, et al. Microsolvation of Sr2+, Ba2+: Structures, energies, bonding, and nuclear magnetic shieldings. Int J Quantum Chem. 2021;121(18):e26753.
- 9. Guan Z, Quinones-Frías MC, Akbergenova Y, et al. Drosophila Synaptotagmin 7 negatively regulates synaptic vesicle release and replenishment in a dosage-dependent manner. Elife. 2020;9:e55443.
- 10. Perez CT, Ferraris J, van Lunteren JA, et al. Adaptive resetting of tuberoinfundibular dopamine (TIDA) network activity during lactation in mice. J Neurosci. 2020;40(16):3203-16.

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