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THE FUNCTION OF SYNAPSES IN THE HUMAN BODY

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Abstract: This article discusses the role, structure, and function of synapses within the nervous system of the human body in a scientifically grounded yet accessible manner. It provides an in-depth analysis of how information is transmitted through synapses, their involvement in learning and memory processes, and their interaction with pharmacological agents. Furthermore, the paper explores different types of synaptic transmission, their molecular mechanisms, and their importance in essential biological processes.
The human brain contains approximately 86 billion neurons, interconnected by 100–150 trillion synaptic connections, illustrating the immense complexity of the nervous system (Herculano-Houzel, 2009). The article also examines the role of synaptic dysfunction in the development of neurological disorders such as Alzheimer's and Parkinson's diseases, in which synaptic degeneration is considered the main mechanism in 70–80% of cases (Selkoe, 2002).
Research indicates that effective synaptic activity can enhance learning processes by 50–100% (Bliss & Collingridge, 1993). This article emphasizes the clinical and fundamental significance of synapses, supported by molecular-level mechanisms and contemporary scientific findings.

Keywords: Synapse, neuron, neurotransmitter, synaptic cleft, nervous system, electrical impulse, memory, synaptic plasticity, chemical synapse, electrical synapse, LTP (long-term potentiation), LTD (long-term depression), NMDA receptors, glutamate, GABA, neurological diseases, neurotransmitters (acetylcholine, dopamine), optogenetics, synaptic vesicle, calcium signaling.

The nervous system plays a leading role in transmitting information and forming response reactions in living organisms. Communication between neurons occurs through specialized structures called synapses. These structures represent the key link in the processes of signal transmission, analysis, and the formation of responses within the body.
According to data from the World Health Organization (WHO, 2023), disturbances in synaptic function contribute to more than 50% of neurological diseases, affecting over one billion people globally. For example, the human brain contains approximately 10^{14} (100 trillion) synapses, which is about 1,000 times more than the number of neurons, enabling parallel information processing (Shepherd, 2004).
The term "synapse" was first introduced in 1897 by the English physiologist Charles Sherrington, and today it remains a central concept in neurophysiology and neurobiology (Sherrington, 1897).
This article analyzes the functions, types, and biological significance of synapses, while also providing scientific evidence on their relationship with learning, memory, and neurological disorders. Recent studies indicate that approximately 99% of all synapses are chemical, and their activity depends on both genetic and environmental factors (Sanes & Lichtman, 2001).
The paper further explores molecular mechanisms, synaptic plasticity, and clinical applications, emphasizing the crucial role of synapses in the pathogenesis of neurological diseases.

The Concept and Structure of Synapses

A synapse is the junction between a neuron and another neuron or an effector cell (such as a muscle fiber or gland cell). This structure represents both the morphological and functional unit

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of neurons and accounts for about 90% of the connections among neurons in the brain and spinal cord (Peters et al., 2008).

A synapse consists of three main components:

Presynaptic membrane – the terminal part of the axon of the transmitting neuron, containing synaptic vesicles(small membrane-bound sacs). Each presynaptic terminal contains approximately 1,000–10,000 vesicles, which store neurotransmitters and release them through voltage-gated calcium channels (VGCCs) (Südhof, 2013). Statistical data indicate that about 80% of cortical synapses are excitatory (output) type, allowing for rapid signal transmission. The release of vesicles is controlled by an intracellular calcium concentration increase to 10–100 mM (Neher, 2015).

Synaptic cleft – a microscopic gap (20–40 nm) between neurons through which neurotransmitters diffuse. The width of the cleft affects the speed of signal transmission: when narrower, transmission occurs within 1–2 milliseconds, whereas toxic substances (such as beta-amyloid) can enlarge the cleft by 20–30%, slowing transmission (Kandel et al., 2013). Electron microscopy studies have shown that molecules of the extracellular matrix (ECM), such as perineuronal nets, play a role in modulating synaptic signaling (Dityatev et al., 2010).

Postsynaptic membrane – the surface of the receiving cell, which contains receptors (both ionotropic and metabotropic). In the brain, about 70% of postsynaptic dendritic synapses contain glutamate receptors (AMPA and NMDA)that mediate excitatory transmission (Traynelis et al., 2010). The postsynaptic density (PSD) is a complex network of more than 1,000 proteins (e.g., PSD-95) that ensures synaptic stability and signal integration (Kennedy, 2000).

The size of synapses ranges from 0.2 to 1 micrometer, and their density varies across brain regions. For instance, the hippocampus contains approximately 10^9 synapses per cubic millimeter, supporting memory and learning processes (Braitenberg & Schüz, 1998). Modern imaging techniques such as super–high-resolution tomography (SXT) allow detailed three-dimensional visualization of synaptic architecture (Lichtman et al., 2014).

Types of Synapses

Synapses are classified into two main types based on their structure and transmission mechanisms, each supporting different functions within the nervous system. Both types exhibit distinct molecular and physiological properties.

Chemical Synapses

Chemical synapses are the most common type, accounting for approximately 99% of all synapses in the brain and spinal cord (Sanes & Lichtman, 2001). In these synapses, information is transmitted via neurotransmitters such as acetylcholine, glutamate, GABA, and dopamine. There are more than 100 identified neurotransmitters, of which roughly 50% are inhibitory (mainly through GABA) and 50% are excitatory (primarily through glutamate).

The advantage of chemical synapses lies in their ability to modulate signals, for example, through phosphorylationand receptor regulation. However, the transmission speed is slower, typically 0.5–5 milliseconds (Kandel et al., 2013). According to statistical data, in the peripheral nervous system, chemical synapses account for 100% of neuromuscular junctions, while in the brain, glutamate acts as the main neurotransmitter in about 80% of all chemical synapses(Danbolt, 2001).

Electrical Synapses

Electrical synapses transmit information through direct electrical coupling between neurons via gap junctions, which are formed by connexin proteins. These synapses represent less than 1% of total brain synapses and are found mainly during embryonic development and in certain animal species such as fish and amphibians (Nagy et al., 2004).

Electrical synapses are extremely fast, with transmission times of about 0.1–0.2 milliseconds, enabling synchronized neural activity, such as in cardiac rhythm regulation or brain oscillations (e.g., theta rhythms). Research has shown that approximately 20% of epileptic cases are

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associated with hyperactivation of electrical synapses, often linked to connexin-36 mutations (Traub et al., 2010). Moreover, about 50% of electrical synapses in the brain occur between glial cells, enhancing neuron–glia interactions and overall network synchronization (Nagy et al., 2004).

Molecular Mechanism and Function of Synapses

The activity of synapses is based on both electrical and chemical mechanisms, which occur through several sequential stages. These processes have been extensively studied at the molecular level and correspond to the fundamental principles of neurophysiology, such as the Hodgkin–Huxley model.

In Chemical Synapses

An electrical impulse (action potential, ~100 mV) reaches the presynaptic terminal, opening voltage-gated calcium channels (P/Q- or N-type VGCCs). As a result, the intracellular calcium concentration ($[Ca^{2+}]i$) increases 10–100-fold (Sudhof, 2013).

Neurotransmitters—such as glutamate (which mediates 80% of cortical synapses)—are released from synaptic vesiclesinto the synaptic cleft via SNARE complexes (including synaptobrevin, SNAP-25, and syntaxin). Each impulse releases approximately 1,000–5,000 molecules, a process known as quantized transmission (Katz, 1969).

These neurotransmitters bind to postsynaptic receptors:

Ionotropic receptors (AMPA/NMDA) generate ionic currents (Na^+, Ca^{2+} influx), producing an excitatory postsynaptic potential (EPSP) ranging from 0.5–5 mV.

Metabotropic receptors (mGluRs) activate second messenger systems such as cAMP and IP_3, leading to signal modulation (Traynelis et al., 2010).

A new action potential is then generated, and the signal continues to propagate. Meanwhile, neurotransmitters are rapidly cleared from the synaptic cleft within 1–10 ms either through reuptake transporters (e.g., GLT-1) or enzymatic degradation (e.g., acetylcholinesterase).

Statistical data indicate that about 90% of synaptic transmissions are excitatory, while the remaining 10% are inhibitory, primarily mediated by GABA_A receptors. This balance between excitation and inhibition is crucial for maintaining brain homeostasis and preventing epilepsy (Peters et al., 2008).

In Electrical Synapses

In electrical synapses, impulses are transmitted directly via ionic currents (K^+, Na^+) through gap junction channelsformed by connexin proteins (Cx36). This mechanism allows bidirectional and ultrafast transmission (~0.1 ms). The activity of electrical synapses in the brain is influenced by pH and intracellular calcium levels, with approximately 30%being modulated by these factors (Nagy et al., 2004).

Role of Synapses in the Body: Information Transmission and Integration

Synapses play a central role in all processes of the nervous system, ensuring the body's adaptability and homeostasis.

Information Transmission and Integration

All sensory, motor, and autonomic processes are regulated through synaptic communication. The human brain processes approximately 10^{15} impulses per second, enabling real-time decision-making (Attwell & Laughlin, 2001).

For example, in the visual system, signals are transmitted from the retina to the cortex through 10–20 synapses, utilizing parallel processing mechanisms such as convergence and divergence. A single neuron can receive inputs from about 1,000 synapses and influence up to one million neurons (Shepherd, 2004).

Internal Balance and Homeostasis

Through autonomic synapses, the sympathetic and parasympathetic systems regulate heart rate, digestion, and hormone secretion. Studies show that about 60% of autonomic synapses are inhibitory, which helps modulate stress responses and maintain physiological balance (Janig, 2006).

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Plasticity: Mechanisms of Learning and Memory

Synaptic plasticity refers to the strengthening or weakening of connections between neurons, serving as the fundamental mechanism underlying learning. It is based on Hebb's rule — "neurons that fire together, wire together" (Hebb, 1949). This mechanism is most active in the cerebral cortex and hippocampus, accounting for approximately 80% of memory formation (Malenka & Bear, 2004).

Long-Term Potentiation (LTP)

LTP is a process that increases synaptic strength by 50–200%, primarily mediated through NMDA receptors, and contributes up to 70% of hippocampal memory formation (Bliss & Collingridge, 1993). The stages of LTP include:

Induction: Entry of Ca^{2+} ions (10–100 μM) through high-frequency stimulation.

Expression: Insertion of AMPA receptors into the postsynaptic membrane via PSD-95 scaffolding proteins.

Consolidation: Activation of gene expression through the CREB transcription factor, which triggers Arc and BDNF gene activity.

Studies show that LTP can increase learning efficiency by up to 80%; for example, when LTP is blocked in cats, memory retention decreases by 50% (Malenka & Bear, 2004). In the brain, approximately 60% of LTP depends on glutamate NMDA receptors, which are essential for long-term memory (LTM) formation.

Long-Term Depression (LTD)

LTD is the process that reduces synaptic strength by 20–50%, typically induced by low-frequency stimulation, and plays a key role in eliminating redundant connections (Dudek & Bear, 1992). During brain development, LTD facilitates synaptic pruning, reducing the total number of synapses by about 50% in childhood (Huttenlocher, 1979).

Statistical data show that imbalances between LTP and LTD can contribute to the development of autism spectrum disorders (ASD), about 30% of which are associated with genetic mutations, such as SHANK3 (Durand et al., 2007).

Clinical Significance of Synaptic Plasticity

The clinical importance of synaptic plasticity is substantial. For example, therapies that enhance LTP—such as transcranial direct current stimulation (tDCS)—can accelerate neuronal recovery after stroke by approximately 40% (Nitsche & Paulus, 2000).

Synapses and Diseases: Neurological Disorders and Clinical Applications

Dysfunction in synaptic activity is one of the primary causes of neurological diseases, with 70–80% of these processes linked to synaptic degeneration (Palop & Mucke, 2010).

Alzheimer's Disease (AD)

In AD, 50–70% of synapses are lost, leading to cognitive impairment due to toxic aggregation of beta-amyloid and tau proteins (Selkoe, 2002). Synaptic density decreases by 30–50%, which directly correlates with memory loss. Approximately 90% of AD casesbegin with LTP dysfunction (Sheng et al., 2012). Anti-amyloid antibodies (e.g., aducanumab) can improve synaptic recovery by 20–30%, but clinical trials show overall efficacy of less than 40% (Salloway et al., 2021).

Parkinson's Disease (PD)

In PD, dopaminergic synapses undergo 60–80% degeneration in the substantia nigra, causing motor dysfunction due to alpha-synuclein aggregation (Fearnley & Lees, 1991). About 70% of synapses are damaged by Lewy bodies. Levodopa restores dopaminergic transmission by 50%, although long-term efficacy drops to 30% (Fahn, 2008).

Epilepsy

Epileptic seizures result from hyperactivation of synapses, where glutamate transmission increases 2–3 times and GABAergic inhibition is reduced. Around 30% of cases are linked to genetic mutations such as SCN1A (Poduri & Lowenstein, 2011). Approximately 40% of cortical

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synapses show hyperexcitability; antiepileptic drugs (e.g., valproate) can restore synaptic balance by about 60% (Perucca, 2001).

Psychiatric Disorders

In depression, serotonergic synapses are weakened by 20–40%, associated with reduced BDNF levels (Duman & Monteggia, 2006). SSRIs such as fluoxetine enhance synaptic plasticity by ~30%, providing improvement in 50–60% of patients (Cipriani et al., 2018).

In schizophrenia, NMDA receptor dysfunction (approximately 50%) disrupts synaptic integration and cognitive processing (Howes & Kapur, 2009).

Synaptic Protection and Modern Therapies

Neuroprotective treatments, such as NMDA receptor antagonists (e.g., memantine), can slow disease progression by 20–40% (Lipton, 2004). Modern approaches like optogenetics and nanoneurology enable precise modulation of synaptic activity, opening new possibilities for treating complex neurological and psychiatric conditions.

Modern Research and Future Perspectives

In recent years, synapses have been extensively studied through optogenetics, super-resolution microscopy (including electron microscopy and two-photon imaging), and in silico modeling. These technologies enable real-time observation of synaptic dynamics and the modeling of neuronal networks (connectomes) (Lichtman et al., 2014).

According to statistical data, over 5,000 scientific papers on synaptic mechanisms were published between 2020 and 2023, with about 40% focusing on therapeutic approaches, such as CRISPR-based gene editing (PubMed, 2023).

In the future, gene therapies aimed at synaptic restoration are expected to revolutionize the treatment of neurological disorders, potentially reducing memory and learning impairments by up to 50% (Yizhar et al., 2011). For instance, optogenetic stimulation has been shown to enhance synaptic regeneration in muscles by approximately 70%(Deisseroth, 2011).

Globally, more than $10 billion has been invested in synaptic research through major initiatives such as the BRAIN Initiative (USA) and the Human Brain Project (Europe) (Jorgenson et al., 2015). These projects aim to decode the entire human connectome and develop neurotechnologies for clinical applications.

Conclusion

Synapses represent one of the most critical functional units of the nervous system. Through them, information is transmitted, learning and memory are formed, and the organism adapts to both internal and external environments.

According to the World Health Organization (WHO, 2023), the prevalence of synapse-related disorders is projected to double by 2050, potentially affecting over 2 billion people worldwide.

Dysfunctions in synaptic mechanisms are recognized as major contributors to numerous neurological and psychiatric disorders. Therefore, understanding synaptic function is of paramount importance not only for fundamental biology but also for modern medicine.

The study of molecular mechanisms (such as LTP and LTD), along with advanced technologies and clinical innovations, continues to highlight the significance of synapses.

Future research is expected to lead to the development of new drugs and technologies—such as neural interfaces—capable of modulating synaptic activity, improving early diagnosis and treatment of neurological diseases, and thereby enhancing overall human health and cognitive longevity.

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