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Brain Neuroplasticity in Students Under Conditions of Intensive Learning: Molecular and Cellular Mechanisms

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Annotation: The objective of the present study is to systematize current knowledge regarding the specific molecular, cellular, and systemic mechanisms of neuroplasticity activated in the brains of students during periods of intensive learning, as well as to identify the endogenous and exogenous factors that modulate the efficacy of these processes.

Keywords:

Introduction

The brain's capacity for structural and functional reorganization in response to experience—known as neuroplasticity—represents a fundamental property of the nervous system that facilitates learning, memory, and adaptation to changing environmental conditions. In recent decades, this concept has evolved beyond a general theoretical framework to acquire specific neurobiological substance: dozens of distinct mechanisms operating at the subcellular, synaptic, neuronal, and systemic levels have been identified and described.

Of particular interest is the investigation of neuroplastic processes in students during periods of intensive learning, when the brain is subjected to sustained cognitive loads associated with assimilating vast volumes of abstract information, developing professional competencies, and preparing for examinations. Gaining an understanding of precisely which neural mechanisms are engaged under such loads not only allows for a deeper insight into the nature of human learning but also enables the development of evidence-based recommendations for optimizing the educational process and maintaining the cognitive health of students.

Research Objective

The objective of the present study is to systematize current knowledge regarding the specific molecular, cellular, and systemic mechanisms of neuroplasticity activated in the brains of students during periods of intensive learning, as well as to identify the endogenous and exogenous factors that modulate the efficacy of these processes.

Materials and Methods

This study was conducted as a theoretical analysis and synthesis of data published in peer-reviewed scientific journals between 2015 and 2025. The review draws upon materials from the PubMed, Scopus, and Web of Science databases, utilizing the following keywords: "neuroplasticity," "synaptic plasticity," "learning," "students," "hippocampus," "neurogenesis," "BDNF," and "myelin plasticity." Particular attention is paid to longitudinal studies employing structural and functional MRI and electrophysiological techniques, as well as to experimental studies using animal models that elucidate underlying cellular mechanisms.

Results and Discussion

1. Synaptic Plasticity: Long-Term Potentiation and Depression

At the cellular level, the key mechanism underlying memory formation is synaptic plasticity, which manifests in two primary forms: long-term potentiation (LTP) and long-term depression (LTD). LTP represents a sustained strengthening of synaptic transmission following high-frequency stimulation, whereas LTD involves the weakening of synapses in response to low-frequency stimulation.

The molecular cascade of LTP is initiated by the activation of postsynaptic NMDA glutamate receptors, leading to an influx of Ca^{2+} ions into the dendritic spine. The resulting increase in intracellular calcium triggers the activity of Ca^{2+}/calmodulin-dependent protein kinase II (CaMKII), which phosphorylates AMPA receptors—thereby increasing their conductance—and facilitates the insertion of new AMPA receptors into the postsynaptic membrane from intracellular reserves. These changes form the basis of the early phase of LTP. To sustain these long-term changes, the activation of transcription factors (e.g., CREB) and the synthesis of new proteins are required, thereby enabling structural rearrangements within the synapse. During periods of intensive learning—such as when memorizing large volumes of terminology or mastering complex concepts—students experience a widespread activation of LTP-like processes within the hippocampus and

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associative cortical regions. Electrophysiological studies indicate that the successful formation of long-term memory correlates with an enhancement of the hippocampal theta rhythm—a frequency range considered optimal for the induction of LTP.

2. Structural Plasticity of Dendritic Spines

Dendritic spines are postsynaptic compartments that serve as the sites for the majority of excitatory synapses. They exhibit remarkable dynamism: in the mature brain, a subset of spines is constantly appearing and disappearing; however, during the learning process, those spines involved in the formation of a memory trace undergo stabilization.

Using *in vivo* two-photon microscopy in animal models, it has been demonstrated that learning motor skills or spatial navigation increases the rate of new spine formation in the corresponding cortical regions and enhances their survival rate. Morphological changes in spines are regulated by the activity of cytoskeletal proteins, particularly actin. Signaling pathways involving small GTPases (RhoA, Rac1, and Cdc42) control actin polymerization, which leads to an enlargement of the spine head and the stabilization of the synapse.

Although direct observation of dendritic spines in humans is not currently possible, high-resolution structural MRI can detect increases in gray matter volume within specific brain regions following intensive learning. For instance, in a classic study by Draganski and colleagues, medical students preparing for an exam over a three-month period exhibited an increase in gray matter volume in the posterior parietal cortex and the hippocampus. These changes likely reflect synaptogenesis, the elaboration of dendritic trees, and glial remodeling.

3. Neurogenesis in the Hippocampus

Contrary to a long-standing dogma, neurogenesis—the formation of new neurons—persists in the brains of adult mammals, albeit confined to two primary regions: the subgranular zone of the hippocampal dentate gyrus and the subventricular zone of the lateral ventricles. In humans, the contribution of adult neurogenesis to cognitive functions remained a subject of debate until recently; however, modern techniques for dating neurons based on their radiocarbon (^{14}C) content have confirmed that hundreds of new granule cells are generated daily within the hippocampus.

New neurons exhibit heightened plasticity and a lower threshold for the induction of LTP, rendering them critically important for certain types of learning—particularly for distinguishing between similar memories (pattern separation). Intensive learning—especially that involving spatial navigation and episodic memory—stimulates the proliferation of neural stem cells and promotes the survival of young neurons. Conversely, chronic stress—often associated with examination periods—suppresses neurogenesis through the action of glucocorticoids. Thus, in students, a complex balance exists between the stimulatory influence of cognitive load and the inhibitory effects of stress.

4. The Role of Brain-Derived Neurotrophic Factor (BDNF)

BDNF (brain-derived neurotrophic factor) is a key regulator of neuroplasticity, serving to link electrical activity with long-term structural changes. It is synthesized within neurons, secreted in an activity-dependent manner, and exerts its effects via the TrkB receptor, thereby triggering intracellular signaling cascades (such as MAPK/ERK and PI3K/Akt) that promote neuronal survival, dendritic growth, and synaptogenesis. Humans exhibit a functional polymorphism of the BDNF gene—a substitution of valine for methionine at codon 66 (Val66Met)—which impairs activity-dependent protein secretion. Carriers of the Met allele demonstrate reduced neuroplasticity, smaller hippocampal volume, and lower scores on certain cognitive tests. In the context of university education, this polymorphism may influence the capacity to assimilate large volumes of information as well as resilience to stress.

Blood BDNF levels correlate positively with cognitive performance and can be elevated through physical activity. Consequently, aerobic exercise is regarded as an effective non-pharmacological method for enhancing neuroplasticity in students during periods of exam preparation.

5. Myelin Plasticity

Traditionally, axon myelination was considered a process that concludes primarily during youth; however, research over the past decade has demonstrated that oligodendrocytes continue to be generated in the mature brain, and myelin can be modified in response to experience. Neuronal activity stimulates the proliferation and differentiation of oligodendrocyte precursor cells, while also modulating the thickness of the myelin sheath surrounding existing axons.

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Myelin plasticity alters the conduction velocity of action potentials and the synchronization of spikes within neural networks—factors that are critically important for cognitive processes. Intensive learning likely enhances myelination in actively utilized tracts—for instance, in the fiber bundles connecting the hippocampus to the prefrontal cortex. In students pursuing complex academic subjects, such microstructural changes in white matter may occur—changes that can be detected using diffusion tensor imaging (DTI).

6. Systemic Reorganization of Neural Networks

At the macroscopic level, learning results in a reorganization of functional connections between different brain regions. The method of resting-state functional MRI allows for the assessment of so-called resting-state networks—large-scale neuronal ensembles that activate synchronously in the absence of a specific task.

In students undergoing intensive learning, functional connectivity undergoes changes within the default mode network (DMN), the frontoparietal network (responsible for cognitive control), and the salience network. Specifically, the successful mastery of material is accompanied by increased connectivity between the hippocampus and the medial prefrontal cortex; this reflects processes of memory consolidation and the integration of new information into existing semantic structures.

7. Modulating Factors: Sleep, Stress, and Physical Activity

The efficiency of neuroplastic processes in students depends significantly on their lifestyle. Sleep plays a critical role in memory consolidation: during slow-wave sleep, the neuronal ensembles that were active during learning undergo reactivation, and synaptic scaling—a global downregulation of synaptic strength—occurs; this process is essential for maintaining homeostasis and preventing synaptic saturation. Sleep deprivation—a common occurrence among students during exam periods—severely impairs LTP and disrupts memory formation.

Through the release of cortisol, stress activates mineralocorticoid and glucocorticoid receptors in the hippocampus; when cortisol levels are chronically elevated, this leads to dendritic atrophy, the suppression of neurogenesis, and the impairment of LTP. However, Conversely, moderate acute stress may facilitate learning by enhancing excitability and attention.

Physical activity increases BDNF expression, stimulates neurogenesis, and improves cerebral blood flow. Meta-analyses confirm the positive impact of aerobic exercise on executive functions and memory in students.

Conclusion

Intensive learning in students triggers a cascade of interconnected neuroplastic processes at various levels of nervous system organization. At the molecular level, NMDA receptors and CaMKII are activated, initiating LTP. At the cellular level, changes in dendritic spine morphology, synaptogenesis, and—potentially—neurogenesis in the hippocampus occur. At the systems level, functional connections between cortical regions are reorganized to facilitate the long-term storage and retrieval of information.

The efficiency of these processes is modulated by endogenous factors (BDNF genotype, hormonal status) and exogenous conditions (sleep, stress, physical activity). Understanding the specific mechanisms of neuroplasticity allows us not only to gain deeper insight into the nature of learning but also to develop evidence-based strategies for supporting students' cognitive functions during periods of peak academic demand. Future research should focus on investigating individual differences and developing personalized approaches to optimize the educational process.

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