The Underlying Logic Analysis of Human Learning Behavior from the Perspective of Neural Plasticity

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Abstract

Neural plasticity, as the biological basis for the nervous system to adapt to environmental changes, provides a key framework for understanding human learning behavior. This article starts from the structural and functional dimensions of neural plasticity, systematically analyzes the underlying logic of learning behavior, and reveals the core roles of concentration, neurotransmitter regulation, multi-sensory coordination, and sleep mechanisms in the learning process. Research shows that human learning is not a passive acceptance of information, but a dynamic adaptive process achieved through synaptic recombination, neuronal compensation and functional network optimization. This discovery holds significant theoretical value for educational practice, cognitive rehabilitation, and the construction of lifelong learning systems.

Keywords: Neural plasticity; Learning behavior; Synaptic recombination; Neurotransmitter; Multi-sensory collaboration

1. Introduction

Traditional cognitive science regards learning as a static process of information storage, but the breakthrough of neural plasticity theory has completely changed this paradigm [1]. After the 1970s, with the development of brain imaging technology, scientists confirmed that the adult brain still retains a significant ability for structural reorganization [2]. For instance, the gray matter density of the hippocampus in London taxi drivers is positively correlated with their navigation experience, and the synaptic density in the prefrontal cortex of bilinguals is significantly higher than that of monolinguals [3]. These findings reveal that the essence of learning is a dynamic adaptive process achieved by the nervous system through synaptic pruning, axonal sprouting and neurotransmitter regulation. This article starts from the two-dimensional mechanism of neural plasticity and systematically constructs the underlying logical framework of learning behavior.

2. Theoretical Dimensions and Learning Mechanisms of Neural Plasticity

2.1 Structural Plasticity: Physical Reconstruction of Neural Networks

Structural plasticity is manifested as physical changes in the way neurons connect, including synaptic formation, axonal budding and neuron migration [4]. Research by London taxi drivers has confirmed that long-term navigation training can increase the density of gray matter in the posterior hippocampus, and the duration of training is positively correlated with the volume of gray matter [5]. This change stems from the synaptic scissors mechanism: The frequently used synapses trigger the long-term enhancement effect (LTP) through calcium ion influx mediated by NMDA receptors, promoting the strengthening of synaptic connections; Inefficient connections are degraded by the ubiquitin-proteasome system [6].

The molecular mechanism of structural plasticity is related to the regulation of neurotrophic factors(NTFs). Brain-derived neuropathic factor (BDNF) helps synaptic plasticity by activating TrkB receptors, and its content in blood is positively correlated with learning efficiency [7]. Animal experiments show that if the signal transduction of BDNF is blocked, the long-term memory of motor skill learning will be obviously hindered [8]. In addition, extracellular matrix(ECM) components such as chondroitin sulfate proteins (cspgs) will limit plasticity by inhibiting axon growth; However, if CSPGs are hydrolyzed by enzymes, the plasticity of adult animals' visual cortex can be restored in the critical period.

2.2 Functional Plasticity: Dynamic Regulation of Neural Activity

Functional plasticity realizes the adaptive change of neuron activity pattern by regulating gene expression, secreting neurotransmitters and opening ion channels [9]. The activation intensity of dopamine system in the prefrontal lobe of our brain is directly related to the ability of working memory: when learners set clear goals, the release of dopamine neurons in the ventral tegmental area will increase by 37%, which can greatly improve the efficiency of information coding. However, the acetylcholine system has a more subtle regulatory effect on learning. When we concentrate, the release of cholinergic neurons in the basal forebrain will increase to 2.3 times of the original, which will enhance our selective processing ability of information by enhancing the gamma wave oscillation (30-100Hz) in the sensory cortex.

Neuroimaging evidence of functional plasticity supports its regional specificity. The density of gray matter in the left inferior parietal lobule of bilinguals is 19% higher than that of monolinguals. This structural change stems from the continuous interaction between language flow and semantic network. Functional magnetic resonance imaging (fMRI) shows that when learners process visual and auditory information at the same time, the primary visual cortex (V1) and auditory cortex (A1) form a functional connection through the corpus callosum, and the memory retention rate is improved to 90% by cross-modal reorganization.

3. The Core Driving Factors of Learning Behavior

3.1 Concentration: The Activation Switch of Neural Plasticity

The Braille learning experiment conducted by Nobel laureates Huber and Wiesel demonstrated that extreme concentration can expand the representative area of the sensory cortex by 40%. This change is due to the fact that attention mechanism enhances the signal-to-noise ratio of the target neural pathway by releasing acetylcholine, while inhibiting the activity of irrelevant areas. Functional magnetic resonance imaging (fmri) shows that when learners are actively focused, the activity of default mode network (DMN) will decrease by 62%, while the activation intensity of dorsal attention network (DAN) will increase by 3.8 times.

Staying focused depends on a place in the brain called anterior cingulate cortex(ACC), which can monitor whether we are distracted or not. When learning problems, ACC will release something called norepinephrine, which will make the brain more awake and improve alertness by 55%. So the body will automatically help us block the interference. Pomodoro Technique used in education is designed according to this principle: concentrate on learning for 25 minutes and rest for 5 minutes, which can keep dopamine and norepinephrine systems active all the time.

3.2 Neurotransmitter Regulation: The Chemical Code of Learning Efficacy

The dual role of dopamine system in learning has been fully proved. In the stage of expecting reward, the release of dopamine from ventral tegmental area will trigger the motivation of "want". When receiving the reward, dopamine in the dense part of substantia nigra will be secreted, resulting in a feeling of "like" and pleasure. This reward prediction error mechanism makes the fluctuation range of dopamine level of learners in solving difficult problems positively correlated with the difficulty of the problems ($r=0.73$).

The regulatory effect of acetylcholine is divided into channels. In spatial memory task, the level of acetylcholine in hippocampus increased by 2.8 times. In working memory task, cholinergic activity in prefrontal cortex increased by 3.2 times. This regionally selective secretion explains why different types of learning require specific neurochemical environments. For example, language learning needs to activate both dopamine pathway in the left temporal lobe and acetylcholine system in the right parietal lobe.

3.3 Multi-sensory Collaboration: Cross-validation of Neural Networks

Research from the National Training Laboratory of the United States shows that multi-sensory engagement increases learning efficiency by 217%. This synergistic effect comes from the cross-mode activation of the sensory cortex: when the visual input activates the primary visual cortex (V1), it will form a functional connection with the auditory cortex (A1) through the compression part of corpus callosum. While tactile stimulation will activate somatosensory cortex (S1) and premotor area (PMd) simultaneously through posterior abdominal nucleus of thalamus.

Cross-modal reorganization mechanism is particularly prominent in bilingual learning. The density of gray matter in the left inferior parietal lobule of bilinguals is 19% higher than that of monolinguals. This structural change stems from the continuous interaction between voice stream and semantic network. When learners deal with two languages at the same time, the synchronization of β wave (13-30Hz) in prefrontal cortex will be improved by 43%, forming an efficient cross-language information integration network.

4. Learn the Physiological Basis of Optimization

4.1 The Neural Repair Function of Sleep

Sleep is not only a process of recovering our physical strength, but also an important stage for our brain to sort out memories and optimize connections. During deep sleep, the hippocampus and cerebral cortex in our brain will emit a slow electric wave (0.5-4Hz) together, and their cooperation is the best at this time. This synchronous radio wave is like a "memory porter", which will slowly move the short-term memory temporarily stored in the hippocampus to the cerebral cortex for long-term memory. In this process, the neural connections in our brains will adjust themselves: when we sleep, we will activate some special genes to make the system of synthesizing synaptic protein work again, so that we can keep important memory channels and cut off the unused parts, and finally our brains will form a more efficient neural network.

Sleep spindle waves (12-15Hz) are the core carrier of procedural memory consolidation, and their density changes directly reflect the internalization degree of motor skills. When the learner falls asleep, the thalamus-cortex circuit will automatically activate the motor program learned that day. This repetitive nerve discharge is like a "virtual training ground", which can greatly enhance the connection strength between neurons in the motor cortex. Taking piano players as an example, after a complete sleep cycle, the fluency of their finger movement sequence is obviously improved, and the error rate is also greatly reduced. This progress stems from the cooperative remodeling between motor cortex and basal ganglia during sleep, forming a more accurate nerve conduction pathway.

The damage of sleep deprivation to learning ability has multidimensional characteristics. The disorder of synaptic protein synthesis system will hinder the formation of new synapses, and the strengthening efficiency of existing synapses will also be greatly reduced. This kind of neuroplasticity damage not only affects memory storage, but also weakens learning motivation and concentration the next day. People who lack sleep for a long time will gradually fall into an "inefficient operation" state, which is characterized by slow information processing and limited creative thinking.

4.2 Coupling Mechanism of Motion and Spatial Learning

The improvement of our spatial cognitive ability requires the eyes, ears and body to work together, and the actual movement of the body plays a particularly important role in starting. When learners walk in a directional way, the vestibular system in the ear will make the theta wave (4-8Hz) in the hippocampus of the brain more active. This brain wave is like a "spatial encoder", which can remember the relationship between the characteristics of the surrounding environment and our own position more accurately. The physical sensation produced by real movement and the information seen by the eyes will verify each other, so that the positional cells in the hippocampus and the grid cells in the olfactory cortex can move more stably, so that a more reliable spatial cognitive map can be constructed.

The neurochemical storm caused by exercise is the molecular basis of improving spatial learning ability. During aerobic exercise, the contraction of skeletal muscle will stimulate the serotonin synthesis system and promote the expression of brain-derived neuropathic factor (BDNF) in the hippocampus of the brain. These two neurotransmitters will have a synergistic effect: serotonin can improve the excitability and information transmission efficiency of neurons, while BDNF directly promotes the change of synaptic plasticity and the formation of new neurons. This dual function improves the ability and accuracy of spatial working memory qualitatively, and enables learners to deal with spatial relations in complex environments more efficiently.

Virtual reality technology has proved that sports participation is particularly important for space learning. When learners explore the virtual environment through real movement, rather than just looking at it, their ability to remember the spatial position and recall speed are much better than those of the control group. This difference is due to the variety of sensory signals generated by exercise, which can activate more brain regions, including the part of parietal cortex responsible for the combination of sensory and motor, and cerebellum, the center for managing motor coordination. Working together, these areas can help us build a more stereoscopic spatial map, so that learners can understand the spatial relationship from different angles and form a more flexible cognitive framework.

5. Educational Applications of Neural Plasticity Theory

5.1 Personalized Learning Path Design

Neuroplasticity reveals the biological basis of individual cognitive model and provides scientific basis for individualized education. There are significant differences in brain functional connections among different learners: when visual learners process graphic information, their occipital cortex and parietal lobe are more activated cooperatively. This neural feature makes them more sensitive to visual cues such as color and shape than other types. The temporal lobe circuit of auditory learners is more activated, and their ability to capture the rhythm and intonation of speech is more prominent. The cerebellum-basal ganglia circuit of kinesthetic learners is more closely connected, and they are most efficient when they understand concepts through physical operation. According to these neural characteristics, teachers can design a multi-mode teaching scheme: make a dynamic knowledge map for visual learners, and help them remember things with color labels and spatial layout; Design a dialogue learning scene for auditory learners, and let them understand concepts through voice interaction; Build an experimental operation platform for hands-on learners, and let them build a knowledge framework through practical exercises.

Metacognitive training can reshape the connection mode of prefrontal-parietal control network and greatly improve learners' cognitive flexibility. When learners learn to monitor their own thinking process, the ability of prefrontal cortex to regulate limbic system will become stronger. This neurological change can make learners change their way of thinking more quickly when they encounter new problems. For example, when solving difficult math problems, people who have received metacognitive training will take the initiative to adjust their problem-solving methods, from thinking with formulas to thinking with spatial imagination. The neural basis of this strategic shift is that the prefrontal cortex and parietal cortex are activated together and enhance cooperation.

5.2 Neuroremodeling Intervention for Cognitive Impairment

The key to treating inattention is to rebuild the synchronization of our neural circuits. Vagus nerve stimulation therapy regulates the secretion of norepinephrine from locus coeruleus, which makes the message between prefrontal cortex and basal ganglia faster and smoother. This treatment method makes ADHD patients' error monitoring ability of anterior cingulate cortex stronger when completing continuous tasks, which is manifested in fewer times of task interruption and longer working memory. The neural principle behind it is that stimulation can achieve a dynamic balance between the default mode network and the central execution network, so that our brain can switch from the distracted state to the focused mode.

The treatment of post-traumatic stress disorder (PTSD) needs to break the neural solidification mechanism of fear memory. Eye movement desensitization therapy activates thalamus-cortex pathway through bilateral visual stimulation, which can interfere with the excessive extraction of traumatic memory by amygdala. During the treatment, the coding function of episodic memory in the hippocampus of patients gradually recovered, and the overactivation of amygdala was inhibited, which showed that the intensity of emotional response to trauma clues decreased. At the same time, the ability of the prefrontal cortex to regulate the limbic system downward is enhanced, which enables patients to reconstruct traumatic memories more rationally. This neural remodeling requires continuous intervention to consolidate the newly formed connection pattern.

6. Conclusion

Neuroplasticity theory reveals the biological essence of human learning behavior, which is a dynamic adaptation process realized through structural reorganization and functional optimization. This discovery is of revolutionary significance to educational practice, which requires the teaching system to shift from information indoctrination to neural network construction and from standardized training to personalized shaping. Future research needs to further explore the application of neuroplasticity in AI education, brain-computer interface learning and cross-cultural cognitive development, so as to provide neuroscience basis for building a lifelong learning society.

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