Memory Reconstruction: A Novel Therapeutic Approach to Psychosis

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Abstract. In the post-COVID-19 era, the rapid changes in the pace of life and the continuous increase in social pressure have led to an escalation in the number of patients with psychosis, which makes the negative impacts at the family and social levels expand. Currently, psychosis patients are mainly healed through medication, psychotherapy, and physical therapy. Most treatment approaches focus on the alleviation of pathological symptoms. However, there is still a deficiency in essential understanding of the more fundamental pathogenic factors and precise abnormal functional regions, thus leaving several gaps in precision medicine. Based on the principle of neural plasticity, this paper elaborates on the neural mechanism of pathological memory and the theoretical basis of memory reconsolidation methods, focusing on the intrinsic mechanisms of mental disorders and pathological memory, and analyzing the deep connection between pathological memory and neural plasticity in psychosis patients. By proposing the conceptual hypothesis of "plasticity threshold", it explores the possible role of memory reconsolidation technology in the treatment of psychosis, providing a reference for the precision medical treatment plan of this technology in such diseases.

Keywords: Psychosis; memory reconstruction; pathological memory; plasticity threshold.

1. Introduction

After the advent of the 21st century, people's living pressure has continuously increased due to the acceleration of lifestyle tempo. Especially within the post-COVID-19 era, changes in the world situation intensified this problem. Due to changes in people's psychological adaptability, some of them will inevitably manifest some psychological symptoms. If these early-onset problems are not detected in time or intervened properly, these can easily develop into psychosis. Current research shows that psychosis is usually associated with some emotional or cognitive function abnormalities and is considered a serious, long-term, and highly recurrent mental disorder. When people are afflicted with this disorder, they usually experience symptoms like delusions, hallucinations, and loss of self-awareness, which can lead to emotional confusion and deviation from reality perception [1]. This undoubtedly seriously interferes with patients' daily life and socialization, and may even lead to malicious behaviors such as autolesion or harming others, bringing unstable factors to both individuals and society. Therefore, it is necessary to deepen the understanding of this issue and combine existing medical means to improve and innovate intervention techniques to minimize the negative impact of the disease to the greatest extent.

Presently, researchers mainly intervene and treat psychosis from three aspects: drugs, psychology, and physics. In terms of drugs, second-generation drugs have made certain breakthroughs in the targeted treatment of specific receptors. Clinical results show [2] that olanzapine can better bind to 5-Hydroxytryptamine (5-HT), Histamine H1, and Dopamine (DA) receptors in the treatment of elderly schizophrenia, affect toxic alkaloid receptors, and selectively act on the mesolimbic DA pathway, having a good intervention effect on emotional, negative and positive symptoms [2]. In psychological intervention, Wang Shanshan et al. [3] effectively intervened in drug abusers through Motivational Interviewing (MI) combined with Cognitive Behavioral Therapy (CBT), and this method was later applied to the intervention of Obsessive-Compulsive Disorder (OCD), achieving good results. Physical methods mainly include brain mechanism research and precise medical treatment. In brain mechanism research, researchers use Resting-state Functional (fMRI) technology

and specific analysis methods to compare first-episode schizophrenia patients with healthy individuals to explore the functional connectivity of brain networks [4]. The study found that patients had reduced functional connectivity within and between multiple brain networks, such as the Default Mode Network (DMN), and connections with higher-order cognitive networks. At the same time, the internal functional connectivity of the Salience network (SC) was reduced, and the subcortical system was disordered, leading to cognitive deficits in patients. In precise medical treatment [5], repetitive Transcranial Magnetic Stimulation (rTMS) as a non-invasive method can regulate neuronal activity and network connections in the treatment of depression, increase the levels of neurotransmitters such as DA and 5-HT in patients, and reduce abnormal miRNA expression, down-regulate the expression levels of miR-18a, miR-124 and miR-214, promote the effective optimization of neurotransmitter synthesis and release, and achieve the effects of improving mood, anti-depression and reducing suicide risk [5].

Although several achievements have been made in the research of the mechanisms and early intervention in psychosis, many key aspects still require in-depth understanding and resolution, such as precision and efficiency. Among them, the complex relationship between psychosis and memory can serve as a breakthrough direction. This article aims to explore the association mechanism between negative memory and mental disorders, and then attempt to intervene in such diseases based on the principle of neural plasticity by using the method of memory reconsolidation.

2. Theoretical Foundation

2.1. Neural Plasticity

Neural plasticity refers to the malleability of neuronal connections and neural circuits. It is essential for the immediate encoding of neural activity, the functioning of critical periods after birth, and the establishment of mechanisms during early development [6]. Therefore, this characteristic is widely recognized as the foundation for an individual's memory, learning, and environmental adaptation. Hebb's learning theory posits that if the pattern of neuronal activity can be self-sustained and the connections between co-activating neurons selectively strengthened, learning and memory can be achieved. This mechanism alters the firing patterns of neurons and regulates the strength of trillions of synapses in the brain [6]. When the presynaptic neuron fires before the postsynaptic neuron, the synapse is strengthened; conversely, if it fires after, the synapse is weakened. When firing is uncorrelated, the strength of the synapse remains unchanged. For instance, when an individual is learning something new, neuronal activity alters the synapses, and these activity patterns are subsequently reinforced unconsciously, forming memories [6]. Research indicates that oxytocin is a substance that plays a crucial role in neural regulation and has the important function of inducing long-term synaptic plasticity. In the CA2 region of the hippocampus, oxytocin can lower the threshold for inducing Long-term Potentiation (LTP) [7]. This makes the neurons in this region more prone to LTP, enabling more efficient encoding and storage of related stimuli, thereby enhancing the individual's learning and memory abilities in social behavior and social cognition. In the auditory cortex, oxytocin can promote LTP induction, activate certain inhibitory neurons, and cause them to release inhibitory neurotransmitters such as Gamma-aminobutyric Acid (GABA). Under specific circumstances, it weakens inhibitory signals, resulting in disinhibition, making neurons more easily activated, and facilitating LTP induction. This mechanism plays a key role in enhancing the response of young organisms to specific sounds, helping them better recognize and respond to certain sound signals, which is crucial for their survival and adaptation [7].

2.2. Neural Mechanisms of Pathological Memory

Current research indicates that specific pathological memories are encoded by sparse neuronal populations. In the study, researchers stereotactically injected the AAV9 virus into the Dentate Gyrus (DG) region of c-Fos transgenic mice. Using optogenetic methods, they detected the expression of ChR2-EYFP and c-Fos through immunohistochemistry and recorded the responses of DG neurons to

light stimulation in vivo, while observing the freezing behavior of the mice to evaluate the recall of fear memory [8]. The experimental results showed that when doxycycline (Dox) was present, DG neurons did not express ChR2-EYFP, but its expression was induced after Dox was withdrawn for 2 days. Moreover, after fear conditioning training, the number of ChR2-EYFP positive cells significantly increased, and they were mainly expressed in excitatory neurons. Regarding the responses of neurons to light stimulation, some DG neurons in mice injected with AAV9-TRE-ChR2-EYFP had reliable responses to light stimulation, while no light-responsive cells were detected in mice injected with AAV9-TRE-EYFP. This indicates that light-induced the recall of fear memory, a phenomenon not observed in the no-shock (NS) group and the EYFP group, thus ruling out the influence of non-specific activation. Therefore, this experiment demonstrates that activating specific hippocampal neuronal populations involved in memory engrams is sufficient to evoke fear memory, and the hippocampus plays a key role in the formation of contextual components of fear memory. The DG is an ideal target for studying pathological contextual memory engrams [8].

2.3. Theoretical Basis of Memory Reconsolidation

Over the past half-century, researchers have investigated the neurobiological mechanisms of memory storage at the synaptic level, finding that it is related to synaptic plasticity, key molecular signaling cascades, and the strengthening of specific synaptic connections. They have also discovered that the hippocampus is an important structure involved in the formation of individual memories and can consolidate long-term memory formation through Long-term Potentiation (LTP) [9]. In molecular mechanism research, researchers have found that short-term memory can be formed by increasing presynaptic glutamate release, altering the activity of postsynaptic glutamatergic receptors, and covalent modification of existing proteins, while long-term memory depends on gene transcription, protein translation, and synaptogenesis [9]. During this process, mitogen-activated protein kinase (MAPK) and protein kinase A (PKA) act together on cAMP response element-binding protein (CREB), and some proteins and genes play important roles at different stages of memory. To explain the persistence of long-term memory in dynamic synaptic connections, researchers have proposed concepts such as calcium/calmodulin-dependent protein kinase II (CaMKII), protein kinase M-zeta (PKMζ), activity-regulated cytoskeletal-associated protein (Arc), and functional prion protein, which have more elaborately described the processes of RNA binding proteins, RNA transport, and epigenetic modifications [9]. Additionally, studies at the cellular level have shown that specific neuronal populations play a key role in memory formation, retrieval, and extinction. These theories suggest that memory reconsolidation can occur at the synaptic level by altering key molecular signaling cascades and the connection patterns of specific synapses to construct a new memory retrieval circuit. After initially establishing this circuit, Deep Brain Stimulation (DBS) can be considered to stimulate the CA3 region of the hippocampus to enhance its ability to integrate and store new memories and form new memory traces. At the same time, new memories can be distinguished from the original ones, and the original pathological memories can be "hidden". In terms of short-term pathological memory, mental disorders such as schizophrenia and depression are characterized by an imbalance between excitation and inhibition. Considering the role of mixed synapses in regulating the excitation-inhibition balance, it is possible to suppress excessive excitation by enhancing the release of GABA in mixed synapses or regulating its effect on AMPA receptors, thereby improving pathological short-term memory. For this purpose, drugs that regulate the function of mixed synapses can be developed to restore normal short-term memory function in patients. Regarding long-term pathological memory, studies have shown that it is possible to intervene in the expression of memory-related proteins by regulating signaling pathways [10]. In the formation of long-term memory, MAPK and PKA [10] act together on CREB. Therefore, for abnormal signaling pathways related to pathological long-term memory, specific inhibition or activation of MAPK or PKA can be considered. For example, patients with schizophrenia may have an overactive MAPK pathway, leading to abnormal expression of certain memory-related proteins. MAPK inhibitors can participate in regulating the phosphorylation level of CREB, affecting downstream gene transcription

and protein translation related to long-term memory, and thereby correcting pathological memory by regulating abnormal signaling pathways [10]. From another perspective, specific neuronal populations [11] also play a key role in memory formation, retrieval, and extinction. Based on this, if optogenetic technology is effectively utilized, it is possible to precisely control the activity of specific neuronal populations related to pathological long-term memory. For instance, for patients with depression, through optogenetic technology, the overactive state of the neuronal population related to negative memories in their bodies can be effectively suppressed. This can inhibit the retrieval of negative memories during the memory retrieval stage, thereby alleviating depressive symptoms.

3. The Association between Pathological Memory and Neural Plasticity

3.1. Neural Representation and Consolidation Mechanism of Pathological Memory

There are significant differences in the neural representation between normal memory and pathological memory. Normal memory relies on the coordinated work of multiple brain regions [11], while pathological memory is more dependent on the amygdala than other brain regions. Therefore, pathological memory is often accompanied by intense negative emotional experiences. Generally, after experiencing trauma, the activity of the amygdala in individuals increases, leading to a significant increase in the intensity and emotional arousal of pathological memory. At the cellular level, traumatic memory also causes high excitation of neurons, thereby altering their plasticity and abnormally activating signaling pathways such as mitogen-activated protein kinase (MAPK) [11]. Compared with the formation process of normal memory, the changes in synaptic plasticity during the formation of pathological memory are more pronounced. This difference makes pathological memory more prone to consolidation. During the consolidation process, the dynamic balance between synaptic plasticity and memory traces is disrupted, resulting in the release of large amounts of glutamate, which activates N-methyl-D-aspartate receptors (NMDAR) and α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptors (AMPAR) on postsynaptic neurons, triggering intracellular signaling cascades, increasing synaptic strength, and possibly forming new synapses, thereby consolidating the traces of pathological memory. In Post-traumatic Stress Disorder (PTSD) [11], when patients are exposed to trauma-related cues, the neural pathways related to traumatic memory in their brains are rapidly activated and difficult to suppress. This process leads to the continuous strengthening of memory traces and their inability to normally fade, thus causing the continuous consolidation of pathological memory in patients with such diseases.

3.2. Bidirectional Regulation Mechanism of Neural Plasticity

The bidirectional regulation of neural plasticity [12] refers to plasticity confinement and plasticity reinitiation, which play a crucial role in the processes of memory and learning. Plasticity confinement typically occurs during neural development, as the neural circuits gradually stabilize, their adaptability and plasticity continuously decrease. This process helps the nervous system maintain established functions and reduces unnecessary changes [12]. In adult individuals, rapid and large-scale reorganization is usually restricted by stable neural connections; plasticity reinitiation refers to the process of neural adaptive changes in which the brain reorganizes its structure, function, or connection patterns after traumatic brain injury [13] to restore lost functions or adapt to new circumstances. This mechanism usually occurs in the context of injury or external intervention. Clinical studies have shown that after stroke or Traumatic Brain Injury (TBI) [13], the brain attempts to reorganize its internal working patterns to restore lost functions as much as possible. Through these two mechanisms, individuals can maximize the balance between the stability required for normal neural functions. Additionally, research has found that neural plasticity is neither transient nor unique to developing organisms [14]. It is generally believed that the nervous system gradually stabilizes and achieves an optimal functional mode as the individual develops, a process that reduces the system's adaptability but does not eliminate it. After the nervous system stabilizes, although plasticity is no longer prominent in neural functions, it still exists and plays a role [14].

Based on the above research findings, some technological innovations can be achieved in the memory reconstruction intervention methods for patients with mental illness. First, researchers can consider developing a Virtual Reality (VR) system in the future, creating immersive environments and gradually exposing patients to stimuli related to pathological memories in a controlled manner to trigger plasticity reinitiation, while using positive or neutral associations to overwrite previously maladaptive memory traces during the process. Secondly, researchers can still consider improving existing optogenetic techniques by targeting specific neural circuits involved in emotion regulation and stimulating neurons in a way that breaks existing, potentially maladaptive neural patterns. Subsequently, combined with cognitive behavioral therapy, individuals can be guided to form new, healthier neural connections, thereby improving emotion-related neural functions.

3.3. Clinical Translation Pathways of Pathological Memory Reconsolidation

Currently, the clinical translation of pathological memory reconsolidation technology can be roughly achieved through the following two pathways. One is to start from a specific molecular circuit model. In experiments with multiple system atrophy as the research object, researchers have deeply understood the relationship between abnormal α-synuclein and memory impairment and found that α-synuclein oligomers may be the pathological cause of memory impairment in multiple system atrophy, leading to memory damage by affecting the structure and function of hippocampal neurons [15]. Therefore, by regulating the related molecular mechanisms to reconsolidate pathological memory, it is possible to intervene in pathological memory abnormalities specific to certain diseases [15]. The other is to continue and improve the bio-psycho-synergistic treatment. Experiments have shown that ATP-dependent chromatin remodeling [16] plays a key role in memory and substance use disorders, especially the memory formation and maintenance mechanisms involved, which are closely related to behaviors such as addiction; Lu Lin et al. developed a non-conditioned stimulus arousal-extinction model [17] based on the regulation of the endocytosis process of ionotropic glutamate receptors in the amygdala brain region in their research. This model can more thoroughly and effectively eliminate all related and long-established pathological memories. All the above findings further confirm that after starting from the molecular mechanism level and combining with psychological intervention techniques, it is possible to achieve effective intervention for pathological memories such as addiction to a certain extent, and through the method of psycho-biological synergy, the technology of pathological memory reconsolidation can be translated into clinical practice.

3.4. Integrated Model: Dynamic Interaction between Pathological Memory and Neural Plasticity

Neural plasticity, as defined and discussed in Section 2.1 of this article, plays a crucial role in the formation, storage, and retrieval of memories. Abnormal neural plasticity can disrupt the neural pathways associated with normal memory. Gilbertson et al. found that the formation of pathological memory during memory consolidation and storage is to some extent related to the structural volume of the hippocampus [18]. Generally, individuals with smaller hippocampal volumes are more prone to developing pathological memory when exposed to traumatic events. Abnormal hippocampal volume often indicates changes in neural plasticity and deviations in the processing of trauma-related memories, which may exacerbate the formation of pathological memory. Additionally, Shin et al. further demonstrated in their study of specific brain regions in PTSD patients that the neural plasticity in brain regions such as the amygdala, medial Prefrontal Cortex (mPFC), and hippocampus is abnormally altered, leading to an imbalance in the integration of emotions and memories and causing excessive flashbacks of traumatic memories in a pathological form [19]. Based on these changes, the concept of a "plasticity threshold" could be proposed in the future. This concept aims to indicate that under normal physiological conditions, neural plasticity is maintained within a certain range, and the memory system is in dynamic balance while the individual's memory function operates normally. However, when individuals encounter pathological events such as trauma or long-term chronic stress, neural plasticity is abnormally altered. If this alteration persists below a specific pathological locking

threshold, the memory system will struggle to return to normal balance and enter a pathological state. Therefore, only when neural remodeling capacity reaches or exceeds this pathological threshold can the adverse effects of pathological memory be alleviated, and the memory system can return to dynamic balance. Thus, memory reconstruction techniques essentially aim to regulate the aforementioned "plasticity threshold" through various intervention methods, balancing this value with the pathological locking threshold to buffer and dissolve pathological memory. Felmingham et al. experimentally demonstrated that cognitive behavioral therapy (CBT) can to some extent alter the functions of the anterior cingulate cortex and amygdala in PTSD patients, guiding them to adjust their cognitive and emotional responses during the process of re-examining traumatic events [20], enhancing the neural plasticity of related brain regions and promoting their development towards exceeding the pathological locking threshold, thereby "reconstructing" the patients' memories to a certain extent; Malberg et al.'s pharmacological research found that some antidepressants can regulate neural plasticity by promoting hippocampal neurogenesis [21]. During the process of neurogenesis, these drugs can alter the release mechanism of neurotransmitters, indirectly enhancing their remodeling capacity and exceeding the "plasticity threshold", facilitating the psychological recovery of patients. Therefore, memory reconstruction can be an effective strategy for addressing pathological memory-induced mental disorders. In the process of efficient implementation, existing methods such as drug therapy, psychotherapy, and physical therapy need to be improved, and a "psychological-biological-physical" intervention system should be formed by exploring deeper relationships to provide more targeted treatment.

4. Current Application Status of Memory Reconstruction

At present, technologies related to memory reconstruction are mainly applied in the treatment of AD and stroke, with relatively few applications in psychosis. In the treatment of AD, memory reconstruction technology can regulate the metabolism of the medial temporal lobe through specific neural techniques, improve the metabolic function of the left hippocampus and the region [22], enhance energy supply, restore the activity of damaged nerve cells and promote signal transmission between neurons. That is based on metabolic recovery, it can re-establish effective memory neural circuits to improve memory function. In addition, this technology can also be used to design targeted cognitive training programs for cognitive training, allowing patients to repeatedly practice memory-related exercises, stimulate the brain to form new neural connections and reconsolidate memory. The combination of these two methods can strengthen the connection between the medial temporal lobe and other brain regions, thereby enhancing the brain's ability to process memory and alleviate the memory decline symptoms of AD patients [22]. In the treatment of stroke, this technology mainly focuses on the recovery of cognitive function. Studies have found that the recovery of cognitive function after stroke is closely related to the increase in functional connectivity of the default network and the decrease in functional connectivity of the salience network. Therefore, memory reconstruction technology can target the default network and salience network, and through specific intervention measures, enhance the functional connectivity of the default network while inhibiting the salience network, accelerating the recovery process of cognitive function [23]. At the same time, combined with technologies such as TMS, it can specifically stimulate brain regions related to the default network to enhance their functional connectivity and promote the recovery of patients' memory function [23].

However, most of the current treatment methods are still focused on mice, and there are still many problems before they can be applied to humans. Moreover, the experimental effects are short-lived and cannot well meet the long-term treatment needs in clinical practice. Although some studies have found an association between brain network reconfiguration and cognitive recovery, the existing technologies still have difficulty in precisely regulating the functional connections of specific regions or networks to achieve efficient and individualized intervention. Therefore, it is difficult to detect the

best timing and methods for early intervention promptly, and it is difficult to fully leverage the technical advantages of precision medicine.

5. Conclusion

There exists a complex and close connection between mental disorders and negative memories. Therefore, an in-depth exploration of the mechanism of their association and the innovation of new treatment concepts are of great significance for the treatment of such diseases. Although this technology has achieved certain results in the mechanism research and early intervention of mental disorders, there are still deficiencies in terms of accuracy and efficiency, and it is still in its infancy. From the current situation, the future treatment of mental disorders should focus on three directions: early diagnosis and treatment, personalized intervention, and precise treatment. In terms of early diagnosis, researchers need to strengthen the research on the identification of early symptoms of mental disorders, integrate psychology and computational science and other fields, and develop more sensitive early diagnosis indicators. For example, by monitoring the subtle changes in neural plasticity in specific brain regions, calculate the early abnormal activities of neural pathways related to pathological memory; in terms of personalized treatment, it can start from the genetic level, screen out the genes that patients may carry diseases, and at the same time combine brain function assessment and psychological state analysis, draw personalized disease maps through a large amount of experimental data, and then design personalized memory reconstruction plans for patients based on the maps, improve the existing cognitive behavioral therapy, and guide patients to form healthy memory and emotional patterns during the process of memory reconstruction; in terms of precise treatment, with the help of neuroimaging technology and molecular biology methods, clarify the specific neural circuit abnormalities and molecular mechanism defects of patients, precisely regulate the signals in the abnormal pathways related to different mental disorder patients, and at the same time use highly specific inhibitors or activators to correct the molecular abnormalities related to pathological memory, achieving precise reshaping of pathological memory. In conclusion, to achieve the healthy development of this technology, it is necessary to improve and enhance the existing technology under the guidance of the above three goals, promote the memory reconstruction technology to achieve greater breakthroughs in the field of mental disorder treatment, reduce the burden on patients and society to a greater extent, and improve the quality of life of patients more efficiently, bringing new hope to more groups suffering from psychosis.

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