Characteristics and neural mechanisms of active forgetting in patients with PTSD

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Abstract. The intrusive thoughts and memory function disorders caused by PTSD have a negative impact on the lives of patients, and the neural mechanism of active amnesia is not yet fully understood. This study aims to explore the characteristics and neural mechanisms of active forgetting in PTSD patients. By studying the characteristics of directed forgetting and suppression-induced forgetting in PTSD patients, as well as understanding and analysing relevant paradigms, behavioural evidence and neural evidence, we finally found that the mechanism of directed forgetting and suppression-induced forgetting in PTSD patients are different. That is, the directional amnesia effect of patients with high-level PTSD is higher than that of patients with low-level PTSD, while the suppression-induced forgetting effect of patients with high-level PTSD is lower than that of patients with low-level PTSD. The reason is that patients with high-level PTSD have a strong sense of self-protection, so they actively avoid emotional materials during the encoding stage of memory, resulting in selective encoding of emotional materials. Therefore, the directed forgetting effect is strong. Individuals with high levels of PTSD have a weakened ability to inhibit emotional material, leading to their inability to stop involuntarily ruminating on it, and thus their suppression-induced forgetting effect is weak. Overall, this study understands the key factors that are beneficial to the recovery of PTSD patients, and at the same time provides certain reference and guidance for the treatment plan of PTSD patients.

Keywords: PTSD, active forgetting, neural mechanism

1. Introduction

PTSD (post-traumatic stress disorder) refers to a mental disorder that results in delayed onset and longterm persistence after experiencing unusually threatening or catastrophic stressful events or situations [1]. It is characterized by disorders of memory and forgetting functions [2], and unconscious or unconscious intrusion of traumatic memories [3]. This has a great impact on the mental health and life of PTSD patients, such as anxiety, work and interpersonal problems. Recent research has begun to focus on the neural mechanisms of active forgetting in PTSD, mainly studying how individuals forget traumatic experiences. Active forgetting refers to the phenomenon of increased forgetting caused by individuals consciously inhibiting the recall of certain memory contents [5], which can occur in the encoding stage and retrieval stage of memory [6]. It helps solve the symptoms of PTSD patients such as continuous, involuntary recurrences and flashbacks of traumatic events. It can also serve as a selfprotection mechanism for PTSD patients and help them alleviate their psychological disorders. However,

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the cognitive neural mechanisms of active forgetting are not yet fully understood. Therefore, this study explored the neural mechanism of active forgetting in PTSD patients when faced with traumatic memories.

The main purpose of this study is to describe the cognitive neural mechanism of active forgetting in PTSD patients by analysing experimental behavioural results and neurophysiological mechanisms in the existing literature. And by understanding the physiological and neural mechanisms of PTSD patients to help them regulate traumatic memories. At the same time, this study will also explore the activation areas of the brain during the active forgetting process and the interactions between brain areas to build a more comprehensive explanatory framework for the active forgetting mechanism of PTSD patients, to more deeply explore how PTSD patients pass through Active forgetting modulates traumatic memories.

Theoretically, this study helps to reveal the key factors in the recovery process of PTSD patients and can also better construct the theory of PTSD patients in coping with traumatic memories by in-depth understanding of the cognitive neural mechanisms of patients' active forgetting. Clinically, the significance of this study is to provide psychotherapeutic workers with specific guidance to help PTSD patients effectively deal with traumatic memories. For example, more personalized and targeted treatment plans can be designed based on the active forgetting mechanism of PTSD patients. These programs not only focus on alleviating traumatic memories, but also include enhancing patients' cognitive memory abilities and emotion regulation strategies to improve overall treatment effectiveness and reduce the risk of relapse.

2. Characteristics and mechanism of active forgetting in patients with PTSD

According to previous research, active forgetting is divided into directed forgetting and suppressioninduced forgetting [5]. Directed forgetting is the phenomenon where memories are impaired due to the disruption of the encoding process, whereas suppression-induced forgetting is the phenomenon where memories are impaired due to suppression during the retrieval phase [7]. Next, this study will elaborate on the characteristics and cognitive neural mechanisms of active forgetting in PTSD patients from these two types of active forgetting.

2.1. Characteristics of directed amnesia in PTSD patients.

The phenomenon of memory impairment caused by inhibition at the encoding stage is called directed forgetting [7]. The study of directed forgetting effects is inseparable from the directed forgetting paradigm. The directed forgetting paradigm mainly examines how people lead to forgetting by blocking and interfering with memory encoding [4]. It is divided into two stages - learning stage and testing stage [10]. According to the different positions where the cue words appear in the learning stage, the directed forgetting paradigm can be divided into item method and word list method. The item method is to present a memory or forgetting prompt word after the subject has read each learning item; while the word list method is to uniformly prompt the subject to remember or forget the previous items after the subject has read a series of items [9]. According to previous research, the generation of directed forgetting effect may be related to five aspects: (1) instructions promote the encoding of words that need to be remembered; (2) instructions inhibit the brain's encoding of words that need to be forgotten; (3) The subjects selectively rehearse the words they need to remember; (4) the subjects selectively rehearse the expected test words; (5) during the test, the subjects selectively search the memory of the words [5]. There are two explanations for the mechanism of directed forgetting. One is the selective encoding theory, that is, each item is in working memory before the cue word to remember or forget appears. After the cue word appears, the memory prompt will cause the subject to encode the item carefully, but the forgetting prompt will guide the subject to clear the item from working memory and not encode it carefully; the other is the inhibitory control theory, that is, a requirement to forget The cue causes subjects to generate positive, inhibitory-related brain activity that prevents further encoding of the item. Previous research reflects that when the project method is used, both theoretical mechanisms are involved in the process of directed forgetting [9].

The characteristics of directed amnesia in PTSD patients derived from previous studies are as follows: PTSD patients have a higher directed amnesia effect for emotional materials than neutral materials; patients with high-level PTSD have a higher directed amnesia effect than patients with low-level PTSD; patients with high-level PTSD have a higher directed amnesia effect than patients with low-level PTSD Active recognition avoidance in the memory processing stage has a greater impact on the directed forgetting effect than the defect of retrieval inhibition [1]. The above study was conducted using the project method and used the TBR - TBF score as an indicator. Among them, the TBR score of emotional materials is higher than that of neutral materials, and the TBF score is lower than that of neutral materials. Therefore, in general, the TBR-TBF score of emotional materials is higher than that of neutral materials. The directed forgetting effect of PTSD patients on emotional materials is higher than that of neutral materials. The TBR-TBF scores of patients with high-level PTSD are higher than those of patients with low-level PTSD, and the TBF scores are lower than those of patients with low-level PTSD. PTSD patients. Therefore, in general, the TBR-TBF score of patients with high-level PTSD is higher than that of patients with low-level PTSD, and the directed amnesia effect is higher than that of patients with lowlevel PTSD. The TBF score of patients with high-level PTSD is significantly lower than that of patients with low-level PTSD. This difference in indicators can be used as a basis for high-level PTSD patients. There is evidence that active recognition avoidance in PTSD patients has a greater impact on the directed forgetting effect during the memory processing stage than the deficit in retrieval inhibition. The reason for reaching the above conclusion is that emotional materials pay more attention to the details of the stimulus during memory processing, negative materials tend to process specific items, and positive materials make subjects pay more attention to the commonality of the stimuli. In the competition for limited psychological resources during the learning process, negative stimuli can receive more attention and thus be processed with priority. When resources are sufficient, positive stimuli are also fully processed. Therefore, different emotional materials receive more attention than neutral materials in memory processing and retrieval; at the same time, high-level PTSD patients can more sensitively identify information that may cause mood swings to them than low-level PTSD patients, and actively block it. This information is used to protect yourself.

2.2. Characteristics of suppression-induced forgetting in PTSD patients

Suppression-induced forgetting is a memory impairment phenomenon caused by inhibition in the retrieval stage [7]. There are two types of suppression-induced forgetting paradigms - direct inhibition and vicarious inhibition. The commonly used paradigm of direct inhibition is the TNT paradigm, which is divided into learning phase, TNT phase and test phase. In the learning stage, subjects were asked to learn "cue-target" word pairs; in the TNT stage, only clue words were presented to subjects. The response condition (think) requires subjects to recall the corresponding target word, and the inhibition condition (no think) is required to avoid recalling the target words. Some of the learned words will not appear in this stage but will be compared with the experimental task words as the baseline level in the test stage; in the test stage, all words presented in the learning stage are tested. If the amount of information recalled under the NT condition is significantly lower than the baseline level, the suppression-induced forgetting effect occurs [12]. Vicarious suppression refers to limiting the brain's retrieval of irrelevant memories by increasing the cognitive resources occupied by dominant ideas. Start by learning cue-target word pairs. In the formal experimental stage, when the clue word is presented, the subject is given an alternative word, and the subject is required to remember the connection between the clue word and the alternative word, which helps the subject not to recall the target word [6]. Some studies have shown that people with high trauma experience have a stronger suppression-induced forgetting effect than those with low trauma experience. Because after experiencing trauma, individuals will continue to suppress the intrusion of traumatic memories, which may enhance the ability of memory suppression. In fact, damage to their memory suppression ability may cause PTSD patients to be unable to remove traumatic memories, and the repressive and amnesic effects are weakened or even eliminated.

There are two explanations for the theoretical mechanism of suppression-induced forgetting, one is the interference theory, and the other is the inhibition theory. Interference is said to be that under the

inhibition condition, the subject mixes the target word with no Think instructs to make connections, causing new connections to be made that compete with the previous memory content, thereby weakening it. There was an increase in memory-related activity; inhibition theory means that suppression is triggered as a direct response, and the subject selectively retrieves the target word. Hippocampal activity decreased during inhibition trials, and memory-related activity decreased. Some studies have shown that the suppression theory is correct, because under suppression conditions, subjects' memory-related activities are weakened rather than enhanced [14]. Some studies have also shown that in the TNT suppression and forgetting paradigm, the interference theory and the inhibition theory are compatible, because interference can capture clues and forget independently in the TNT paradigm. The difference in the hippocampus between the recall condition and the NT condition may reflect the enhanced activation under the recall condition, rather than the suppression of memory under the NT condition. Explanation of this relative difference depends on whether learning competition theory is used to process the data or inhibition theory [15].

2.3. Neural mechanism of active forgetting

According to previous research, the mechanism of suppression-induced forgetting in ordinary people is as follows: (1) As the intensity of repeated suppression of intrusions continues to decrease, the activation of the ventral attention network also decreases. At the same time, during the inhibition process, the functional connections between the executive control network increased significantly, the functional connections within the default network and visual cortex decreased, and the functional connections within the dorsal attention network increased [3]; (2) ALFF in a large number of frontal lobe areas related to executive control (Mainly including dorsolateral prefrontal lobe, supplementary motor area, superior frontal gyrus, etc.) and Reho (right dorsolateral prefrontal lobe, supplementary motor area, and orbitofrontal lobe) are positively correlated with active forgetting ability [6]; (3) dlPFC (dorsolateral prefrontal cortex area) and HC have opposite neural activity patterns under memory suppression conditions [6]; (4) Gray matter in the dorsolateral prefrontal area (DLPFC/BA9) and anterior cingulate location (ACC). There is an obvious negative correlation between volume and individual inhibition ability [4]; (5) The hippocampus, a memory-related brain area, and the amygdala, an emotional processing brain area, are inhibited [4]. This matches the reduced memory-related activity noted in the behavioural evidence, thus supporting the suppression theory as an explanation for the amnestic effect of suppression. The neural evidence of PTSD in previous studies mainly focused on suppression-induced forgetting, and there is a lack of neural evidence of directed forgetting. The neural mechanism of suppression-induced forgetting in PTSD patients is different from that of ordinary people. Patients with PTSD exhibit a different neural mechanism for suppression-induced forgetting compared to the general population. Their parietal-occipital-temporal network shows higher gamma energy under the 'no think' condition than under the 'think' condition. This implies that individuals with PTSD have an increased amount of recall under the 'no think' condition, and the effect of suppression-induced forgetting is diminished. [1]. Because the amount of recall is lower than the think condition and baseline level, the suppression-induced forgetting effect occurs. This is also consistent with the evidence mentioned above that the impairment of memory suppression ability may cause PTSD patients to be unable to remove traumatic memories. And the activity of the left middle frontal gyrus on the right side is lower than that of ordinary people [16], and the higher the WBSI score, the lower the activity of the left middle frontal gyrus on the right side. This shows that PTSD patients have higher WBSI scores than ordinary people. They suffer from uncontrollable intrusive thoughts and have weak suppression-induced forgetting effects. Additionally, increased amygdala activity explains the acquisition, maintenance, and expression of conditioned fear memories. The disinhibition of the amygdala by the medial prefrontal cortex is one of the reasons that promotes the emergence of PTSD. At the same time, the excitability of the frontal lobe is reduced. As mentioned earlier, the frontal lobe area is positively correlated with the ability to actively forget. That is, the stronger the neural activity in the frontal lobe, the stronger the ability to actively forget. Therefore, its ability of suppression-induced forgetting is weakened [13].

3. Discussion

The difference between directed forgetting and suppression-induced forgetting is that the former is a memory impairment phenomenon caused by inhibition in the memory encoding stage, and the latter is a memory impairment phenomenon caused by inhibition in the retrieval stage. Moreover, the directed amnesia effect of high-level PTSD patients is higher than that of low-level PTSD patients, while the suppression-induced forgetting effect of high-level PTSD patients is lower than that of low-level PTSD patients. This is because patients with high-level PTSD are more sensitive to emotional materials, and their strong self-protection motivation causes them to avoid memorizing emotional materials during the learning stage, distracting their attention, and selectively encoding emotional materials. Therefore, patients with high-level PTSD have a high forgetting effect in the encoding stage of memory, that is, a high directed forgetting effect. As for suppression-induced forgetting, patients with high-level PTSD have already memorized the material, and their poor inhibition ability makes them involuntarily recall the material. Therefore, patients with high-level PTSD have a weak forgetting effect in the memory retrieval stage, that is, a weak suppression-induced forgetting effect. The connection between directed forgetting and suppression-induced forgetting is that they both involve inhibition theory, that is, the subject generates inhibitory brain activity after receiving relevant instructions, thereby achieving the purpose of forgetting.

The shortcoming of existing research is that there is a lack of neural evidence for directed forgetting in PTSD patients, such as which brain areas' activities and connections are related to directed forgetting in PTSD patients, and how the activity and connections in these brain areas are different from ordinary people. At the same time, the sample size was small due to the limited number of people with high PTSD levels three and a half years after the earthquake. Only 70 samples that met the requirements were screened out. It would be more appropriate to increase the number to 100. And some studies did not exclude additional variables, such as subjects' depressive state scores. This may affect the subjects' feelings when seeing negative words and thus affect the active forgetting effect. And only neutral materials were used instead of emotional materials for research, such as studying the relationship between inhibitory ability and grey matter volume, which lacks generalizability. Because the addition of emotional materials will make the entire study more comprehensive and convincing. This is because it is not clear whether the conclusions obtained by using emotional materials are consistent with those obtained by using neutral materials.

4. Conclusion

This study summarizes active forgetting and its types and paradigms based on previous research. At the same time, it compares the neural mechanism of active forgetting in PTSD patients with ordinary people and summarizes the characteristics of active forgetting in PTSD patients: high degree of PTSD. The directed forgetting effect of patients was higher than that of patients with low-level PTSD, while the suppression-induced forgetting effect of patients with high-level PTSD was lower than that of patients with low-level PTSD. This is due to the different mechanisms of directed forgetting and suppression-induced forgetting in PTSD patients. Patients with high-level PTSD are more sensitive to emotional materials, and their strong self-protection motivation causes them to avoid memorizing emotional materials. And its poor inhibition ability makes it involuntarily recall the material. The significance of this study is that it can help better understand the pathogenesis of PTSD and develop effective treatment plans to help PTSD patients alleviate symptoms such as flashbacks, recurrences, and hypervigilance, so that they can better integrate into social life.

5. Prospects

According to the shortcomings of existing research, in the future, we can study the neural mechanism of directed forgetting in PTSD patients by exploring the activities and connections of brain areas related to directed forgetting in PTSD patients, to clarify their differences from ordinary people, and to understand their pathogenesis and treatment methods. The sample size can also be increased in future

studies. A feasible method is to change the sample to a larger group. And excluding additional variables such as depression status scores makes the experimental results more accurate. At the same time, emotional materials are used to study the relationship between inhibition ability and gray matter volume, to clarify whether the conclusions of research using emotional materials and neutral materials are consistent, and to enhance the generalization of the research.

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