

The Influence of Traumatic Memories in Schizophrenia

Jingyi Feng

UFR IHSS – Faculty of Societies and Humanities, Paris Cité University, Paris, 75340, France
Corresponding author. Email: fengjingyi_zhengya@163.com

Abstract : Since studying psychiatric disorders, Clinicians have observed and reported the abnormal nature of the presence of traumatic memories and the main symptoms they cause in PTSD, an essential aspect of which is memory encoding. The leading cause of traumatic memory flashbacks is the inability to store certain representations of the traumatic event in the hippocampus due to deficits in the hippocampus. Hippocampal deficits are also found in schizophrenia, and some studies suggest that the hippocampus is the central core of the neuropathology of schizophrenia. In contrast, sleep spindles play a vital role in sensory processing and long-term memory consolidation. Studies collected in the field of PTSD suggest that sleep spindles can alleviate mood. However, schizophrenia patients commonly have sleep disturbances and significantly reduce spindles and sleep-dependent memory consolidation. So, in conclusion, it can be speculated that mood swings cause memory encoding problems that the sleep spindles waves cannot handle.

Keywords: traumatic memories, schizophrenia, PTSD, memory encoding, sleep spindles.

1. Introduction

Among people with traumatic memories and schizophrenia, data suggest that people with traumatic experiences are more likely to develop schizophrenia. PTSD is a trauma-induced disorder, and sleep disturbances are prevalent in schizophrenia and PTSD, as well as impairments in memory encoding in the brain. According to research, childhood trauma is associated with memory impairment and reduced hippocampal volume in adult survivors [1]. However, in a study of patients with PTSD, research has demonstrated that some traumatic events cannot be stored in declarative memory; instead, they exist at the level of sensorimotor and affective, being organized in the implicit memory of the amygdala [2]. In other words, the patient will not be able to store the traumatic event in his or her memory and then translate it but will only experience the emotional experience repeatedly.

For people with schizophrenia, traumatic memories are an essential factor. Childhood trauma is a form of stress associated with impaired working memory, language learning, and attention in people with schizophrenia [3]. On the other hand, in a seminal study, between an experimental group of people with schizophrenia, dissociative disorder patients, and non-illness phantom listeners, there were no groups significant differences in the form of experiencing hallucinations [4]. The hallucinatory forms experienced by the patient and non-patient groups were similar, and the differences between the groups were mainly related to the content of the sounds, emotional quality, and control points. This study suggests that hallucinatory forms in disorders such as schizophrenia are closely related to traumatic memories.

In order to measure variability across individuals, the psychological community has proposed the diathesis-stress model to explain the causes of mental illness [5]. This model suggests that different psychological or physical qualities have different capacities to withstand environmental stress and that individuals have different thresholds for breakdown. Once this latch is exceeded, it can lead, for example, to the onset of illness [6,7]. In this paper, the primary purpose is to explore how memory problems caused by traumatic memories can compress this neural model in schizophrenic patients. The effect of traumatic memories on sleep spindles waves reveals that schizophrenic patients commonly have sleep disturbances and that spindles and sleep-dependent memory consolidation are significantly reduced in schizophrenic patients [8]. In contrast, in PTSD patients, it is known that sleep spindles waves play an essential role in memory consolidation in response to anxiety after exposure to stress.

2. Traumatic Memory and Memory Encoding

Among the symptoms caused by traumatic memories, it has been frequently discovered that traumatic memories are distinguished by discontinuity, strong sensory influences, and a lack of verbal narrative content. In order to measure the degree of trauma in a more detailed way, Bessel A van der Kolk et al. proposed the Traumatic Memory Scale (TMI) to systematically evaluate the memory organization, which includes: traumatic experience, the mode of recovery, changes in post-traumatic memory characteristics over time, and differences in memory between subjects with and without current PTSD [9]. Regardless of the age at which they experienced trauma, all traumatized patients in this study claimed that their trauma returned in the form of somatic sensations and emotional flashbacks. With greater intensity, the traumatic memories flash back in consciousness and activate more modes of sensation along with the affective component. From this, it can be concluded that traumatic experiences are, to some extent, recreated in the human soma. According to empirical evidence from neuropsychological studies [2,10,11], the leading cause of traumatic memory flashbacks is due to deficits in the hippocampus. Studies of memory functioning in PTSD patients consistently show that certain representations of traumatic events cannot be stored in (via hippocampal functioning) declarative or narrative memory; they are initially organized only at the sensorimotor and emotional level in the so-called implicit, procedural memory (based on the amygdala). In another word, information assessed by the amygdala is converted into emotional and hormonal signals that trigger emotional responses when it is transmitted to the systems controlling behavioral and neurohormonal responses in the brainstem, so that people may have their emotional systems hormonally activated before they can respond consciously to their reactions [12]. In an EEG study [13], increased lateralization and activation of the right hemisphere were evident in people under the influence of trauma. When traumatic memories are reactivated, however, the brain re-experiences the traumatic memory but cannot translate this experience into communicable language. Namely, the patient may have a similar situation of "indescribable fear," and their body may react as if they were back at the time of the experiences traumatic; their body reacts accordingly.

After the emotional system in the right half of the brain has been activated, other brain structures further respond to this information. These include the hippocampus, which begins to organize and categorize this information with previously existing information that resembles sensory input. The strength of hippocampal activation is positively correlated with the strength of amygdala input; the more significant the meaning given by the amygdala, the stronger the input received and the better the memory retained. In a study on animals [14,15], high levels of stimulation of the amygdala disrupted hippocampal function. Such high levels of emotional activation may significantly disorder the correct assessment and distribution of experiences by interfering with hippocampal function. Van der Kolk hypothesized that when this happens, the sensory imprint of the experience will be stored

in memory. However, these different imprinting cannot be fully unified into a whole due to the impaired integration of the hippocampus [11].

2.1. Affection of Traumatic Memories to PTSD & Schizophrenia

It can conclude that traumatic experiences are usually imprinted as sensations or sensory states and are not gathered and produced into personal chronicles. Traumatic memories will not be stored in the hippocampus but the amygdala. Similarly, studies have shown a 12% reduction in left hippocampal volume in 17 children with maltreatment-related PTSD compared to 17 case-matched controls in the PTSD population [16]. These findings suggest that reduced left hippocampal volume is associated with maltreatment-related PTSD. In this study, adults with child maltreatment-related PTSD had significantly reduced hippocampal volume bilaterally compared to non-maltreated controls. However, this reduction was not seen in children with maltreatment-related PTSD. The hippocampus of children with maltreatment-related PTSD was intact; hippocampal atrophy occurred sometime between childhood and adulthood following the abuse. Such a study may prove that memory causes hippocampal atrophy rather than damage at the time. It is also consistent with previous research that "high levels of emotional arousal may interfere with hippocampal function and significantly disrupt the correct assessment and categorization of experiences." These information follows those high levels of emotional arousal in the amygdala interfere with the storage function of the hippocampus and prevent memory encoding, which later leads to hippocampal atrophy due to the continued inability to encode memories.

In schizophrenia, the hippocampus occupies the same important position. It has been concluded in the literature that the hippocampus is a significant core of the neuropathology and pathophysiology of schizophrenia[17], and there is much evidence pointing to a link between schizophrenia and the hippocampus, as shown by magnetic resonance imaging (MRI) studies that patients have reduced hippocampal volume, altered hippocampal shape, and that smaller hippocampal volumes were already present in subjects with the first episode of the illness, suggesting that the reduced hippocampal volume is not a result of side effects of the illness or its subsequent treatment[18-20].

In addition, hallucinations, as a common symptom of schizophrenia, are also closely associated with traumatic memories. In a study of past traumatic memories and hallucinations in people with schizophrenia [21], volunteers who heard voices often felt a connection between the voice and the traumatic experience. Relational associations were the most common, with up to 50% of the association between voices and the trauma perpetrator. One study found that those who advertise their hallucinations as intrusive and controlling are more likely to have experienced similar subjects of trauma [22]. Meanwhile, a more intuitive study found that 33% of people claimed to have experienced hallucinations directly associated to past trauma, with the remaining 67% reporting hallucinations related to their trauma. [23]. Based on these results, it is possible to identify "somatic sensations" and "emotional flashbacks" in schizophrenia similar to those seen in PTSD. It has been suggested that these hallucinatory contents are affected because the cognitive-emotional circuits encoded in episodic memory appear to reappear in the auditory experience [24]. According to Hardy et al., this sense of threat may lead to some negative judgments about self, other people, and the world. These judgments may form the content of the sound hallucination concerning past interactions with others [22].

So, it is easy to see those memory encoding problems due to traumatic memories account for a considerable proportion of the symptoms of schizophrenia. Due to prefrontal and hippocampal dysfunction, declarative memory impairment is known to lead to cognitive impairment in schizophrenia. According to the research of Kathrin Zierhut et al. in their research investigation, schizophrenic patients, irrespective of successful or unsuccessful memory encoding, showed hippocampal hyperactivity during deep encoding, as well as the anterior cingulate cortex (ACC) and dorsomedial prefrontal cortex (DMPFC) activity was reduced but left inferior frontal cortex (LIFC)

activity was regular, directly linking the memory deficit to dysfunctional hippocampal hyperactivity[25]. The activity level of the sleep spindle wave also seems to confirm that memory encoding problems are responsible for the above symptoms.

2.2. Traumatic Memories Affect Sleep Spindles

The sleep spindle wave is an EEG marker of non-rapid eye movement (NREM) sleep. It is a 12 - 16hz oscillation generated by the thalamic reticular nucleus (TRN) interacting with other thalamic nuclei. It is then transmitted to the cortex via the thalamus-thalamus and thalamus-cortex [26]. Research on sleep spindle waves confirms that sleep spindle waves play a crucial role in sensory processing and long-term memory consolidation [27]. In addition, sleep spindle activity, which integrates new information into existing knowledge systems, has been associated with improved procedural and declarative memory [28]. In this study, the researchers divided the volunteers into two groups: the awake group and the sleep group. Both groups were given information to remember. The members of the sleep group were given pieces of information before the sleep, opposite to the awake group. It is worth noting that sleep spindle activity was associated with nocturnal lexical integration in the sleep group but not with recall rates or speed of recognition of new words. Thus, sleep seems to play an active role in integrating new words in information through spindles activity. In contrast, during school learning, the learning dependence of sleep spindle density increases during the nighttime sleep period following a learning session [29]. It was demonstrated that sleep affects memory consolidation, while the practice of memory tasks, in turn, affects the structure of post-training sleep [30].

The study by Nikhilesh Natraj et al. highlights that NREM sleep can regulate anxiety after exposure to stress in PTSD and has a vital role in memory consolidation [31]. Not only does it alleviate emotions, but memory consolidation may be affected when fear reminders are present during slow-wave sleep after abrogating memory learning [32]. However, at this stage, it is impossible to conclude that sleep spindle waves can prevent the onset of PTSD due to limitations, but an increased peak frequency of sleep spindle waves can be observed in patients [33]. In a study of schizophrenia and the sleep spindle, patients with schizophrenia showed a significant reduction in the spindle and sleep-dependent memory consolidation, suggesting, in part, that a significant reduction in the sleep spindle may be causally related to schizophrenia [8]. Furthermore, it has been shown in numerous studies that patients with schizophrenia have sleep spindle deficits and that spindle deficits predict cognitive decline—also, reduced amplitude and sigma power of the individual spindle correlated with the severity of positive symptoms. An abnormal state of the sleep spindle impairs sleep-dependent memory consolidation in schizophrenic patients, leading to positive symptoms; this could be an essential part of treating cognitive deficits in schizophrenia.

Sleep disturbances have been present in psychotic patients since Bleuler's first clinical observations of schizophrenic patients. Recent evidence suggests that sleep disturbances can lead to psychotic symptoms, including delusions and hallucinations [34]. As observed in sleep electroencephalography (EEG) [35], atypical brain connectivity and plasticity are central characteristics of schizophrenia pathology [36]. For example, sleep disturbances in schizophrenia, especially in the acute phase of psychosis, occur with severe insomnia and fragmented, restless sleep. As mentioned above, the sleep spindles play an essential role in procedural memory, and Tamminen J's study demonstrated that sleep could improve memory structure [28]. However, Manoach DS's study found no significant improvement in MST (Motor sequence task) performance after sleep in schizophrenia. The relationship between spindles activity and clinical symptoms is solid [37,38], with several studies on sleep showing that: sleep spindles deficits are negatively associated with psychiatric severity in patients with schizophrenia, including chronic schizophrenia, and notably, nine

adolescents comply with the criteria for early-onset schizophrenia, they also reduced sleep spindle activity [39].

In summary, both hallucinations and memory deficits in schizophrenia can be attributed to problems in memory encoding and, in the case of PTSD patients, to high levels of amygdala stimulation that interfere with hippocampal function and cannot be translated into language. From the study of the sleep spindles wave, it can be assumed that mood swings affect memory encoding during sleep and that when the mood swings exceed the processing range of the sleep spindles wave, the information is not stored and remains "emotional."

3. Conclusion

Many scholars have researched traumatic memory and memory encoding in these studies, which discussed the impact of traumatic memories on patients with schizophrenia, all more or less revealing the impairments in memory encoding caused by traumatic memories and correlating these impairments with the symptoms presented by the patients. However, it is still challenging to define "traumatic memory." Once a "memory" has been encoded in the brain, it is inevitably distorted, whether it is a traumatic or standard memory. At the same time, much research in the field of schizophrenia suggests hippocampal dysfunction in schizophrenic memory encoding deficits and memory encoding problems in schizophrenic patients. However, there is a lack of evidence that traumatic memory encoding problems are the leading cause of some symptoms in schizophrenic patients. The degree of activity of the sleep spindles wave can, to some extent, corroborate the mechanisms by which traumatic memories operate in schizophrenic patients, and the similarities between the pathological manifestations of PTSD patients and schizophrenic patients can be linked to the argument. However, PTSD cannot be equated with traumatic memories, and research has shown that PTSD symptoms are associated with more significant emotional distress but not schizophrenia-specific symptoms [40]. However, both PTSD patients and schizophrenia patients share similar presentations in terms of memory storage problems. So, to some extent, this can be used as a reference. In the future, the understanding of how traumatic memory encoding affects people with schizophrenia and the role of the sleep spindles wave, could lead to position about the role of sleep in treating or preventing schizophrenia.

References

- [1] McCabe, Kathryn L et al. "Relationship between childhood adversity and clinical and cognitive features in schizophrenia." *Journal of psychiatric research* vol. 46,5 (2012): 600-7.
- [2] Van der Kolk, B.A., Filler, R. Dissociation and the fragmentary nature of traumatic memories: Overview and exploratory study. *J Trauma Stress* 8, 505–525 (1995).
- [3] Popovic, David et al. "Childhood Trauma in Schizophrenia: Current Findings and Research Perspectives." *Frontiers in neuroscience* vol. 13 274. 21 Mar. 2019.
- [4] HONIG, ADRIAAN M.D., Ph.D., MRCPsych.1; ROMME, MARIUS A. J. M.D., Ph.D.2; ENSINK, BERNARDINE J. M.Sc., Ph.D.2; ESCHER, SANDRA D. M. A. C.2; PENNINGS, MONIQUE H. A. M.A.2; DEVRIES, MARTEN W. M.D., Ph.D.2 Auditory Hallucinations: A Comparison between Patients and Nonpatients, *The Journal of Nervous & Mental Disease*: October 1998 - Volume 186 - Issue 10 - p 646-651
- [5] APA Dictionary of Psychology. dictionary.apa.org. [2019-03-22].
- [6] Lazarus R. S. From psychological stress to the emotions: A history of changing outlooks (PDF). *Annual Review of Psychology*. 1993, 44 (1): 1–21 [2019-03-29].
- [7] Sigelman, C. K. & Rider, E. A. (2009). *Developmental psychopathology. Life-span human development* (6th ed.) (pp. 468-495). Belmont, CA: Wadsworth Cengage Learning.
- [8] Erin J. Wamsley, Matthew A. Tucker, Ann K. Shinn, Kim E. Ono, Sophia K. McKinley, Alice V. Ely, Donald C. Goff, Robert Stickgold, Dara S. Manoach, *Reduced Sleep Spindles and Spindle Coherence in Schizophrenia: Mechanisms of Impaired Memory Consolidation?*, *Biological Psychiatry*, Volume 71, Issue 2, 2012, Pages 154-161

- [9] Van der Kolk B, Hopper J Exploring the nature of traumatic memory: Combining clinical knowledge with laboratory methods *Journal of Aggression, Maltreatment and Trauma*, Osterman J *Journal of Aggression, Maltreatment and Trauma*, (2001), 9-31, 4(2)
- [10] Brewin, C. R. (1998). Intrusive autobiographical memories in depression and post-traumatic stress disorder. *Applied Cognitive Psychology*, 12, 359–370
- [11] Bessel A van der Kolk (1994). *The Body Keeps the Score: Memory and the Evolving Psychobiology of Posttraumatic Stress* January 1994 *Harvard Review of Psychiatry* 1(5):253-65
- [12] LeDoux, J. E. (1992). Brain mechanisms of emotion and emotional learning. *Current Opinion in Neurobiology*, 2, 191-197.
- [13] Scott L. Rauch ; Bessel A. van der Kolk; Rita E. Fisler; et al. A Symptom Provocation Study of Posttraumatic Stress Disorder Using Positron Emission Tomography and Script-Driven Imagery, *Arch Gen Psychiatry*. 1996; 53(5):380-387.
- [14] Adamec, R. E. (1991). Partial kindling of the ventral hippocampus: Identification of changes in limbic physiology which accompany changes in feline aggression and defense. *Physiology and Behavior*, 49, 443-453.
- [15] Squire, L. R., & Zola-Morgan, S. (1991). The medial temporal lobe memory system. *Science*, 153, 2380-2386.
- [16] Bremner, J D et al. "Magnetic resonance imaging-based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse--a preliminary report." *Biological psychiatry* vol. 41,1 (1997): 23-32. doi:10.1016/s0006-3223(96)00162-x.
- [17] Harrison, P.J. The hippocampus in schizophrenia: a review of the neuropathological evidence and its pathophysiological implications. *Psychopharmacology* 174, 151–162 (2004).
- [18] Lawrie S, Abukmeil S (1998) Brain abnormality in schizophrenia. A systematic and quantitative review of volumetric magnetic resonance imaging studies. *Br J Psychiatry* 172:110–120
- [19] Nelson MD, Saykin AJ, Flashman LA, Riordan HJ (1998) Hippocampal volume reduction in schizophrenia as assessed by magnetic resonance imaging—a meta-analytic study. *Arch Gen Psychiatry* 55:433–440
- [20] Wright IC, Rabe-Hesketh S, Woodruff PWR, David AS, Murray RM, Bullmore ET (2000) Meta-analysis of regional brain volumes in schizophrenia. *Am J Psychiatry* 157:16–25
- [21] Van den Berg, David, et al. "Voice Phenomenology as a Mirror of the Past." *Psychological Medicine*, 2022, pp. 1–9
- [22] Hardy, A., Fowler, D., Freeman, D., Smith, B., Steel, C., Evans, J., ... Dunn, G. (2005). Trauma and hallucinatory experience in psychosis. *Journal of Nervous and Mental Disease*, 193(8), 501–507
- [23] Peach, N., Alvarez-Jimenez, M., Cropper, S. J., Sun, P., Halpin, E., O'Connell, J., ... Bendall, S. (2020). Trauma and the content of hallucinations and post-traumatic intrusions in first-episode psychosis. *Psychology and Psychotherapy: Theory, Research and Practice*, 94, 1–19.
- [24] Brewin, C. R., Gregory, J. D., Lipton, M., & Burgess, N. (2010). Intrusive images in psychological disorders: Characteristics, neural mechanisms, and treatment implications. *Psychological Review*, 117(1), 210–232.
- [25] Kathrin Zierhut, Bernhard Bogerts, Björn Schott, Daniela Fenker, Martin Walter, Dominik Albrecht, Johann Steiner, Hartmut Schütze, Georg Northoff, Emrah Düzel, Kolja Schiltz, The role of hippocampus dysfunction in deficient memory encoding and positive symptoms in schizophrenia, *Psychiatry Research: Neuroimaging*, Volume 183, Issue 3, 2010, Pages 187-194
- [26] Huguenard JR, McCormick DA. Thalamic synchrony and dynamic regulation of global forebrain oscillations. *Trends Neurosci*. 2007;30(7):350–356.
- [27] Muller, Lyle; Piantoni, Giovanni; Koller, Dominik; Cash, Sydney S; Halgren, Eric; Sejnowski, Terrence J (2016-11-15). Skinner, Frances K (ed.). "Rotating waves during human sleep spindles organize global patterns of activity that repeat precisely through the night"
- [28] Tamminen J, Payne JD, Stickgold R, Wamsley EJ, Gaskell MG (October 2010). "Sleep spindle activity is associated with the integration of new memories and existing knowledge". *The Journal of Neuroscience*.
- [29] Schmidt, Christina et al. "Encoding difficulty promotes postlearning changes in sleep spindle activity during napping." *The Journal of neuroscience : the official journal of the Society for Neuroscience* vol. 26,35 (2006): 8976-82. doi:10.1523/JNEUROSCI.2464-06.2006
- [30] Maquet, P., Laureys, S., Peigneux, P. et al. Experience-dependent changes in cerebral activation during human REM sleep. *Nat Neurosci* 3, 831–836 (2000).
- [31] Nikhilesh Natraj, T.C. Neylan, L.M. Yack, T.J. Metzler, S.H. Woodward, S.Q. Hubachek, C. Dukes, N.S. Udupa, D.H. Mathalon, A. Richards Sleep spindles favor emotion regulation over memory consolidation of stressors in PTSD
- [32] Ai S-Z, Chen J, Liu J-F, He J, Xue Y-X, Bao Y-P, et al.. Exposure to extinction-associated contextual tone during slow-wave sleep and wakefulness differentially modulates fear expression. *Neurobiol Learn Mem*. (2015) 123:159–67. 10.1016/j.nlm.2015.06.005

- [33] Dan Denis, Ryan Bottary, Tony J. Cunningham, Shengzi Zeng, Carolina Daffre, Kaitlyn L. Oliver, Kylie Moore, Samuel Gazecki, Augustus Kram Mendelsohn, Uriel Martinez, Karen Gannon, Natasha B. Lasko and Edward F. Pace-Schott « Sleep Power Spectral Density and Spindles in PTSD and Their Relationship to Symptom Severity » *Front. Psychiatry*, 19 November 2022 Sec. Psychopathology
- [34] Reeve S, Sheaves B, Freeman D. The role of sleep dysfunction in the occurrence of delusions and hallucinations: A systematic review. *Clin Psychol Rev.* 2015;42:96–115.
- [35] Sprecher KE, Ferrarelli F, Benca RM. Sleep and plasticity in schizophrenia. *Curr Top Behav Neurosci.* 2015;25:433–458.
- [36] Stephan KE, Friston KJ, Frith CD. Dysconnection in schizophrenia: from abnormal synaptic plasticity to failures of self-monitoring. *Schizophr Bull.* 2009;35(3):509–527.
- [37] Ferrarelli F, Peterson M, Sarasso S, Riedner B, Murphy M, Benca R, Bria P, Kalin N, Tononi G. Thalamic Dysfunction in Schizophrenia Suggested by Whole-Night Deficits in Slow and Fast Spindles, *American Journal of Psychiatry*, 2010 P1339-P1348, 167(11)
- [38] Fabio Ferrarelli, M.D., Ph.D., Michael J. Peterson, M.D., Ph.D., Simone Sarasso, Ph.D., Brady A. Riedner, B.A., Michael J. Murphy, Ph.D., Ruth M. Benca, M.D., Ph.D., Pietro Bria, M.D., Ned H. Kalin, M.D., and Giulio Tononi, M.D., Ph.D. Thalamic Dysfunction in Schizophrenia Suggested by Whole-Night Deficits in Slow and Fast Spindles Published Online: 1 Nov 2010
- [39] Tesler N, Gerstenberg M, Franscini M, Jenni OG, Walitza S, Huber R. Reduced sleep spindle density in early onset schizophrenia: a preliminary finding. *Schizophr Res.* 2015;166(1–3):355–357.
- [40] Resnick, S. G., Bond, G. R., & Mueser, K. T. (2003). Trauma and posttraumatic stress disorder in people with schizophrenia. *Journal of Abnormal Psychology*, 112(3), 415–423.