Advances in Social Science, Education and Humanities Research, volume 561
Proceedings of the 2021 2nd International Conference on Mental Health and Humanities Education (ICMHHE 2021)

The Neural Mechanism of Memory Encoding

Qiwen Zhang[1,a,†,*] Yahua Huang[2,b,†,*] Min Yan[3,c,†,*]

[1] *School of Psychology, Lancaster University, Bailrigg, Lancaster LA1 4YW, United Kingdom*
[2] *School of Psychology, University of Washington, Seattle 98105, Washington, United States*
[3] *School of Electronic Information Engineering, Beijing Institute of Technology, Beijing, Beijing, 100081, China*
[*] *Corresponding author. Email: [a]zhangq32@lancaster.ac.uk, [b]huang438@uw.edu, [c]1120182652@bit.edu.cn*
[†] *These authors contributed equally.*

ABSTRACT
Memory encoding is a part of the process of memory. It is the initial stage of learning information and encoding them from different sensory inputs into the brain. Much previous research has demonstrated the crucial role of memory encoding for Alzheimer's patients and schizophrenia patients in cognition. However, most research was focused on memory retrieval, especially from the perspective of working memory and implicit memory. Our review aims to identify the role of memory encoding by reviewing studies of the hippocampus and amygdala in the medial temporal lobe (MTL), also applying several animal studies and then discuss how interventions can improve memory encoding with changes of neural mechanism. However, most literature reviews in the past only focus on memory encoding itself, ignoring its communication with other mental activities. Most experiment designs in previous research utilize cross-sectional studies and only concentrate on a single hippocampal intervention, which makes them unable to afford a whole picture of hippocampal memory encoding. Future research could pay more attention to investigating the connections between memory encoding and cognitive abilities, and improve the current cognitive training to help people suffering from Alzheimer's or other diseases with cognitive impairment.

Keywords: *Memory Encoding, Medial Temporal Lobe, Hippocampus, Amygdala, Medial Prefrontal Cortex, GABAergic interneurons, Animal Studies*

1. INTRODUCTION

Memory can be defined as a process of encoding, storing and retrieving information in an individuals' mind [1]. Cubitt suggested that memory was an important part of the mental equipment which allows individuals to adapt to social settings [2]. Memory encoding is the initial learning of information. It is a process of changing information coming from sensory input into a form then it can be stored in the brain [3]. There are three main types of encoding: visual, acoustic and semantic encoding. A previous study demonstrated psychotic major depression was mainly associated with deficits in encoding and other cognitive impairments [4]. Another study of Alzheimer's disease suggested that patients had impaired visual stream that affects their late stage of visual encoding [5]. The evidence showed the importance of investigating memory encoding because it can help people who are suffering from these kinds of cognitive recognition.

Most of the previous research in cognitive psychology focuses on memory retrieval, especially from the perspective of working memory and implicit memory. Working memory refers to short-term storage of information which allows for the manipulation of the stored information like decision-making and reasoning [6]. A key step in the process of working memory is retrieving information from long-term memory [7]. Previous studies on their relationships suggested that individuals with high working memory capacity were more able to choose and use appropriate retrieval strategies to generate cues by themselves than individuals with low working memory capacity who respond randomly [8]. Another research area is implicit memory. It refers to the influence of unconscious recollection of prior knowledge on current experience and behaviours [9]. The most extensively investigated manifestation of implicit memory is priming, a technique in which the introduction of one stimulus influences the subsequent stimulus. Previous work showed that there were reduced or even eliminated priming effects in meaning properties

than physical features, suggesting that implicit memory is used independently of semantic-level retrieval [10].

From the perspective of working memory and implicit memory, we have learned about memory retrieval. Working memory helps us to retrieve useful information from our long-term memory. For implicit memory, previous experience or unconsciousness will have an impact on our current behaviours. Memory encoding is irreplaceable since it is the starting stage of memory. There are many disorders having the problem of memory encoding. Hence, it is quite meaningful to make a further study on memory encoding. However, research regarding memory encoding is limited, because there is a gap in the past recognition. We will review memory encoding studies from the following three aspects: neuroimaging, animal model and intervention. This review aims to fill the gap, which will also give some directions for future study.

2. ABNORMAL MEMORY ENCODING MECHANISM

The medial temporal lobe (MTL) includes several structures, such as the hippocampal region and amygdala. Over the past years, many researchers have demonstrated the importance of MTL in human and animals' memory systems especially for episodic memory [11]. Episodic memory is a type of long-term memory and refers to recollecting memory of specific events and experiences. However, the role of MTL in memory encoding is not clear as the relationship between MTL and memory. Epilepsy is a disorder marked by sudden recurrent episodes of sensory disturbance and abnormal electrical activity results in events called seizures. Temporal lobe epilepsy (TLE) originates in the temporal lobe of the brain and is associated with impaired episodic memory. The functional imaging of healthy individuals has shown material-specific activation patterns, the left hemispheric activation for verbal memory encoding [12]. Sidhu et al. studied the patients with TLE due to unilateral hippocampal sclerosis (HS) and aged 19 to 56, 24 of them were in right (RHS) and the others were in the left (LHS). They also investigated healthy individuals aged between 19 to 58 [13]. All participants needed to do the verbal and visual memory test while their brain images were being scanned. By comparing the brain imaging of RHS patients, LHS patients and healthy individuals, different medial and lateral temporal lobe activations with the frequency seizure in LHS and RHS patients during face and word encoding were presented. Patients with shorter duration of epilepsy, later age at onset and lower seizure frequency would have less disruption of the memory encoding because it was associated with the memory activations in brain regions that were involved in successful memory formation [13]. Findings suggested that MTL was associated with memory encoding of human cognition.

A previous study showed MTL is crucial for the cognitive process of memory encoding. Emotion also is a part of memory, based on McGaugh's view, the amygdala to the declarative memory system within MTL is critical for consolidating memory for events aroused by emotions [14]. Amygdala is medially within the MTL and is a primary role of emotional responses. Murty et al. analysis indicated that human beings and animals have consistent bilateral activations in the amygdala during successful encoding of emotional memories [15]. The impairment of the amygdala was correlated with a disrupted MTL-dependent memory [14]. Together with other findings of Sergerie et al., suggesting that the MTL-amygdala interactions have a key role in human emotional memory encoding [16].

Moreover, declarative memory, also known as explicit memory, referring consciously recalling facts and events. Declarative memory disturbances have been known as the main cognitive impairment in schizophrenia. It has previously been attributed to the functional and structural abnormalities in the prefrontal cortex as well as the hippocampus [17]. Zierhut et al. aimed to explore whether hippocampal dysfunction contributes to schizophrenia-related encoding deficits and whether this effect is related to positive symptoms of schizophrenia patients [18]. Positive symptoms include hallucinations, delusions and repetitive movement that are hard to control. They investigated patients with paranoid-hallucinatory schizophrenia and the control group. The researchers used fMRI to assess schizophrenia patients' and normal vision individuals' brains when they were encoding words in semantic judgment and case judgment tasks. Evidence suggested that patients' memory impairment came along with a specific pattern of hippocampal activation. Left hippocampal activity was increased during deep encoding (semantic judgment). The declarative memory impairment was correlated with positive symptoms of patients, and positive symptoms were associated with the left hippocampal activation in infrequently occurring events of successful semantic encoding [18]. This demonstrated that the overactive abnormal activities of the hippocampus were related to an impairment of memory encoding for schizophrenia patients.

Recent research on memory encoding in Alzheimer's disease by applying Functional Magnetic Resonance Imaging (fMRI) measures the blood flow of healthy normal elderly participants and patients with Alzheimer's disease when they were doing the three conditions ("novel"; "repeat"; "baseline") with colour photographs of indoor and outdoor scenes [19]. Researchers demonstrated that Alzheimer's disease patients showed impaired explicit memory. However, their implicit memory has not been impacted by the disease [19]. The fMRI brain images also presented patients' mesial temporal lobe were not activated when they were doing the novel versus repeated scenes comparing with the

healthy normal individuals [19]. This suggested the abnormal activations in response to novel scenes and normal reductions inactivation for repeated relative to novel scenes in the mesial temporal lobe and is correlating with the memory encoding of explicit memory for Alzheimer's disease patients. The findings of fMRI also suggested a dissociation in Alzheimer's disease between intact implicit memory in the earlier-stage occipital cortex and impaired explicit memory encoding in the mesial temporal lobe [19]. Therefore, explicit memory and implicit memory are associated with different brain areas for encoding memory.

3. MEMORY ENCODING IN ANIMAL MODEL

Schema in cognitive psychology refers to a collection of basic knowledge about a single concept. When our brains encode information different from established prior knowledge, this new information can be assimilated into a related schema you have established previously and thereby expand the schema's knowledge base. Memory assimilation into schema is an important part of associative memory, the activation of memory that occurs by the recall of something associative with it. Previous studies on the neural mechanism of associative memory encoding suggest that the assimilation of new paired-associate memories relies on the hippocampus and slow cortical learning systems, and is distinct from this fast hippocampal learning system [20]. More recently, Takeuchi's experiments with rats challenge this concept and further demonstrate that hippocampal-dependent new paired-associates learning is associated with a remarkable regulation of immediate early genes expression in the prelimbic region of the medial prefrontal cortex, and that pharmacological interventions targeted at the prelimbic region can prevent both new information and the recall of remotely or even recently consolidated learning [21]. To figure out whether the prelimbic region of the medial prefrontal cortex is involved in memory encoding, rats were trained with a certain schema and then divided into four groups: no conditions, retrieve the original schema, exposed to new paired-associate information, exposed to new non-associated information. The group exposed to new paired-associate information has the highest immediate early gene expression in the prelimbic region, showing that the prelimbic region is activated when assimilating new paired-associate information into previously established schemas during the hippocampal-dependent process. To further prove the role of the prelimbic region in the medial prefrontal cortex played in memory encoding, rats were treated and grouped as before but this time their prelimbic regions were inactivated by blocking AMPA or NMDA receptors. Blockage of either AMPA or NMDA receptors impaired rats' memory consolidation, further verifying the necessity of cortex during assimilation of new paired-associate information into an existing cortically based schema. These findings indicate that schema-dependent memory encoding not only depends on the hippocampus but also has links to cortex encoding.

In addition to schema-dependent memory encoding, episodic memory is another kind of associative memory. Different from schema-dependent memory which focuses on the influence of established prior knowledge and concepts on current encoding, episodic memory focus on the recollection of specific events and situations in the past. Similar as the schema-dependent memory encoding, episodic memory encoding requires the participation of both hippocampus and cortex. Previous studies suggest that the real-time episodic memory needs neurogranin translation in the synaptic compartment of the hippocampus [22]. And the long-term episodic memory encoding requires excitatory medial prefrontal cortex neurons [23]. Bero performed optogenetic inhibition of excitatory medial prefrontal cortex (mPFC) neurons during contextual fear conditioning and reported that temporally precise inhibition of the mPFC inhibited activation of the entorhinal-hippocampal circuit and impairs the formation of long-term episodic memory [22]. To figure out the effect of mPFC on long-term episodic memory, they used contextual fear conditioning to investigate the mechanism behind long-term memory encoding. During contextual fear conditioning, mice learn an association between a novel context and an event like foot shock that occurs in that context. The results show that fear conditioning significantly increases immediate early gene expression in the mPFC. To further investigate how mPFC influences long-term episodic memory, they used an in vivo optogenetic approach to permit temporally precise manipulation of excitatory mPFC neurons in freely behaving mice. Separate groups of mice were injected with either a third-generation halorhodopsin (eNpHR3.0) fused to enhanced yellow fluorescent proteins (EYFP) or with EYFP vector alone, implanted with a fiber optic in the mPFC, received continuous optogenetic inhibition of mPFC excitatory activity during contextual fear conditioning, and tested their recent as well as remote long-term memory. Results show that compared with EYFP controls, mice expressing eNpHR3.0-EYFP which results in mPFC-specific expression spent significantly less time during both the recent and the remote memory tests, suggesting that optogenetic inhibition of excitatory mPFC neurons during memory encoding impairs long-term memory formation. In addition to the research on long-term episodic memory, Jones's experiment in de novo protein synthesis shows that real-time contextual memory formation requires experience-dependent translation of neurogranin in the synaptic compartment of the hippocampus and its 3'UTR interaction with fragile-X mental retardation protein [23]. Generally speaking, real-time episodic memory encoding requires neurogranin translation in the synaptic compartment of the hippocampus and long-term episodic

memory encoding requires excitatory medial prefrontal cortex neurons.

Previous studies indicate that activity in the hippocampus increased when retrieving recollection of the learning episodes consciously [24]. Many studies also demonstrate that hippocampal cells contribute to memory encoding about the perceptual and behavioural structure of experience [25]. It is obvious that the hippocampus participates in both memory retrieval and memory encoding. But whether memory retrieval and memory encoding use the same or separate systems in hippocampus was still unknown. Paulsen and Moser explored the potential role of hippocampal GABAergic interneurons in providing spatial and temporal conditions for modifications of synaptic weights during transference between hippocampus-dependent memory processes [26]. Against the previous assumption that different parts of the hippocampus play different roles, they suggest that the same cells can participate in both encoding and retrieval but with different compartments involved and mediated by GABAergic interneurons. During memory encoding, GABAergic interneurons inhibit hippocampal principal cells' axonal conduction but action potentials can backpropagate to hippocampal principal cells' dendrites. If the backpropagating action potentials coincide with incoming signals, synaptic weights might be modified in the synapses to store information as long-term memory. During memory retrieval, backpropagation and synaptic plasticity are reduced by GABAergic neurons while the axonal transmission is now allowed, and information stored in the synapses can be accessed and transferred to downstream areas. Hippocampal GABAergic interneurons participate in information processing by providing spatial and temporal conditions for modifications of. In a word, hippocampal transitions between the encoding and retrieval modes are controlled by GABAergic interneurons that selectively target axonal or dendritic cell compartments.

4. INVENTION OF MEMORY ENCODING

Reinforcement learning is mostly about relevant experiences and general preferences, while episodic memory is typically based on one's specific and previous experienced events. Reinforcement can enhance memory encoding. Hippocampus will release dopamine in the process of reinforcement learning, which is regarded to offer reward prediction errors signals (RPE). When human and non-human animals get experience and preference, they will be motivated. Then dopamine will produce RPE signals, which help to enhance memory encoding. In that case, the more one is motivated by or interested in some information, the more likely that he or she will memorize them. A study was carried out by Jang et al. to see how RPE made an impact on memory encoding [27]. For the first experiment, it included learning and memory tasks. There were three phases in learning tasks: value, image and feedback. Participants would learn the amount of reward in the value phase. Then the image phase would appear, giving participants a chance to choose "play" or "pass". There were three possibilities here. If one chose "play" and get the reward, he or she would get the score. But if one chose "play" and the trial was not rewarding, he or she would lose 10 points. If the participant chose "pass", the score would remain. After the image phase, they would see a feedback trial and see their score. In the first experiment, the possibilities of animate and inanimate images were oppositely yoked. The possibilities would be calculated then. For memory tasks, participants were given both "old" images and "new" images. They were asked to select old ones and rated their confidence. They also made a hierarchical regression model to support their suppose. In the second experiment, they separated the reward rate from RPE. Participants could not know the probabilities. The rest were very much similar to the first experiment. We could get the results after these two experiments. We could see that rewards and image RPEs play an important role in memory encoding. And that in the current paradigm, the result is precise and consolidated. As rewards are getting more, people would be more likely to devote themselves to the test and to memorizing the image. Then memory encoding would be improved compared to groups that have fewer rewards. In conclusion, rewards do help to enhance memory encoding.

Memory encoding can be enhanced through computerized training. In recent years, fMRI technology has offered possibilities for us to see how our brain changes during the training process. By observing brain changes, we can know whether if memory encoding has been improved. Also, the improvement of intervention can last for months in some areas of our brains, which are also the areas that are responsible for attention and learning. These provide plasticity for memory encoding's improvement through training. An experiment was carried out by Wiest et al. They used a pre-test/ post-test design in students' daily school life in order to verify computerized cognitive training's positive effect. Students who had learning-related delays took part in the experiment. There are Verbal and Symbolic tasks for working memory. And Finger Windows and Number Letter tasks for visual sequence. Each student needed to complete Verbal and Symbolic tasks and Finger Windows and Number Letters in the form of games. Students first did the training and then received pre/post-tests. When students took part in the training, they would earn points according to their accuracy of the performance. Since the program was adaptive, everyone could make progress in their own pace. The training lasted for 20 hours and was included in the schools' schedule. Every student accepted the same amount of training and subtests. As for the result, visual encoding had a small effect while verbal encoding produced an obvious improvement. There were

improvements in both visual and verbal encoding compared to previous performances when subjects have not received training. This project shows the feasibility of improving memory encoding with the help of training.

We have known about the training effect, but whether memory encoding can make an impact on the neural mechanism remains unknown. From the passage above, we have learned that the hippocampus plays a role in both memory encoding and retrieval. The transitions of Hippocampal GABAergic interneurons have different impacts on the memory retrieval process, thus regulating other neurons. Although encoding and retrieval systems are closely connected, whether specific training can enhance the memory encoding system is still not clear. To get the conclusion, Dale et al. have conducted a study. They used MEG skills to get high gamma-band oscillation (HGO) of auditory processing (AT) and computer games (CG). By doing cognitive training, neuromodulation is produced, thus improving cognition. MEG can examine the intervention-related differences during memory tasks. Subjects did AT and CG tasks before and after training and the results were recorded by MEG. Subjects who had suffered schizophrenia were randomly separated into AT group and CG group in the parent trial. AT included auditory processing exercises while CG contained computer games. After the same amount of exercising, subjects were asked some questions and their total intervention time was recorded. In the parent trial, enjoyable results were reported in both trials. Researchers got better MEG data quality and better accuracy from subjects than not receiving the training. When they used MEG, a time-frequency-optimized spatially adaptive filter was also utilized to prevent auditory sources' mislocalization. The test was done during MEG's using, so these changes have not occurred in retrieval. As for the result, they found that no matter in General Recognition or in VLM, there were different improvements in AT and CG. In the left-hemisphere, AT could enhance HGO during the process of auditory stimulus encoding. In the meantime, CG could enhance HGO in the right-hemisphere in stimulus encoding. Both AT and CG can bring an impact on the neural mechanism of memory encoding. This result indicates that training could indeed alter memory encoding's neural mechanism.

5. LIMITATION AND FUTURE DIRECTION

Some limitations in previous research should be noted here. Most literature research we review above only focuses on the neural mechanism of memory encoding. The intercommunication between memory encoding and other mental abilities is still unknown. Without understanding the relationships between them, the investigators are unable to control the intervention of other mental abilities on memory encoding. In addition, their experiments are limited to cross-sectional studies. This kind of study can reveal a general model concluded from a large number of representatives but it cannot show a complete and developing process. More longitudinal studies should be conducted in the future. The long-term tracking of systematic and regular research on the same object can help us see the whole process and key changes in the the hippocampal memory encoding. To further understand the role of hippocampus in memory encoding, future researchers are supposed to investigate the hippocampal circuit of memory encoding and other mental abilities and the circuit of hippocampal interventions affecting memory encoding.

6. CONCLUSION

From the passages above, several aspects of memory encoding were reviewed. We could see that the hippocampus and amygdala in MTL play a major role in emotional and cognitive memory encoding. Both schema-dependent memory encoding and episodic memory encoding rely on the hippocampus and prefrontal cortex. And hippocampus can complete the transfer between encoding and retrieval with the help of GABAergic interneurons' mediation. There are ways to improve one's memory encoding, for example, rewards and computerized cognitive trainings. Despite these conclusions we get, we have found some places that need to improve. Just as we mentioned above, previous research lacks the investigation on connections between memory encoding and cognitive abilities, tracing study and effects of a combination of multiple interventions on memory encoding. In the future, computerized training may be put into children's schedules, through which their memory encoding can be enhanced. Or perhaps future research can apply cognitive training to Alzheimer's treatments to see if doing so can repair their neural mechanism, thus helping them to restore cognition. There are many more applications waiting for us to explore.

REFERENCES

[1] Squire, L. R. (2009). Memory and brain systems: 1969–2009. Journal of Neuroscience, 29(41), 12711-12716.

[2] Cubitt, G. (2013). History and memory. Manchester University Press.

[3] Craik, F. I., & Rose, N. S. (2012). Memory encoding and aging: a neurocognitive perspective. Neuroscience & Biobehavioral Reviews, 36(7), 1729-1739.

[4] Kelley, R., Garrett, A., Cohen, J., Gomez, R., Lembke, A., Keller, J., Reiss, A. & Schatzberg, A. (2013). Altered brain function underlying verbal memory encoding and retrieval in psychotic major

depression. Psychiatry Research: Neuroimaging, 211(2), 119-126

[5] Golby, A., Silverberg, G., Race, E., Gabrieli, S., O'Shea, J., Knierim, K., Stebbins, G. & Gabrieli, J. (2005). Memory encoding in Alzheimer's disease: an fMRI study of explicit and implicit memory. Brain, 128(4), 773-787

[6] Cowan, Nelson (2008). What are the differences between long-term, short-term, and working memory?. Prog. Brain Res. Progress in Brain Research. 169. pp. 323–338. doi:10.1016/S0079-6123(07)00020-9. ISBN 978-0-444-53164-3. PMC 2657600. PMID 18394484.

[7] Cowan, N., Towse, J. N., Hamilton, Z., Saults, J. S., Elliot, E. M., Lacey, J. F., & Hitch, G. J. (2003). Children's working-memory processes: A response-timing analysis. Journal of Experimental Psychology: General, 132, 113–132.

[8] Unsworth, N., Brewer, G. A., & Spillers, G. J. (2012). Working memory capacity and retrieval from long-term memory: the role of controlled search. Mem Cogn, 242–254.

[9] Graf, P., & Schacter, D. L. (1985). Implicit and explicit memory for new associations in normal and amnesic subjects. Journal of Experimental Psychology: Learning, memory, and cognition, 11(3), 501.

[10] Schacter, D. L., Chiu, C. Y. P., & Ochsner, K. N. (1993). Implicit memory: A selective review. Annual review of neuroscience, 16(1), 159-182.

[11] Squire, L. R., Stark, C. E., & Clark, R. E. (2004). The medial temporal lobe. Annu. Rev. Neurosci., 27, 279-306

[12] Golby, A. J., Poldrack, R. A., Illes, J., Chen, D., Desmond, J. E., & Gabrieli, J. D. (2002). Memory lateralization in medial temporal lobe epilepsy assessed by functional MRI. Epilepsia, 43(8), 855-863.

[13] Sidhu, M. K., Stretton, J., Winston, G. P., Symms, M., Thompson, P. J., Koepp, M. J., & Duncan, J. S. (2015). Factors affecting reorganisation of memory encoding networks in temporal lobe epilepsy. Epilepsy Research, 110, 1-9.

[14] McGaugh, J. L. (2004). The amygdala modulates the consolidation of memories of emotionally arousing experiences. Annu. Rev. Neurosci., 27, 1-28.

[15] Murty, V. P., Ritchey, M., Adcock, R. A., & LaBar, K. S. (2010). fMRI studies of successful emotional memory encoding: A quantitative meta-analysis. Neuropsychologia, 48(12), 3459-3469

[16] Sergerie, K., Lepage, M., & Armony, J. L. (2006). A process-specific functional dissociation of the amygdala in emotional memory. Journal of Cognitive Neuroscience, 18(8), 1359-1367.

[17] Heckers, S., Rauch, S., Goff, D., Savage, C., Schacter, D., Fischman, A., & Alpert, N. (1998). Impaired recruitment of the hippocampus during conscious recollection in schizophrenia. Nature neuroscience, 1(4), 318-323.

[18] Zierhut, K., Bogerts, B., Schott, B., Fenker, D., Walter, M., Albrecht, D., ... & Schiltz, K. (2010). The role of hippocampus dysfunction in deficient memory encoding and positive symptoms in schizophrenia. Psychiatry Research: Neuroimaging, 183(3), 187-194.

[19] Golby, A., Silverberg, G., Race, E., Gabrieli, S., O'Shea, J., Knierim, K., ... & Gabrieli, J. (2005). Memory encoding in Alzheimer's disease: an fMRI study of explicit and implicit memory. Brain, 128(4), 773-787

[20] Lisman, J. E., & Grace, A. A. (2005). The hippocampal-VTA loop: controlling the entry of information into long-term memory. Neuron, 46(5), 703-713.

[21] Tse, D., Takeuchi, T., Kakeyama, M., Kajii, Y., Okuno, H., Tohyama, C., ... & Morris, R. G. (2011). Schema-dependent gene activation and memory encoding in neocortex. Science, 333(6044), 891-895.

[22] Bero, A. W., Meng, J., Cho, S., Shen, A. H., Canter, R. G., Ericsson, M., & Tsai, L. H. (2014). Early remodeling of the neocortex upon episodic memory encoding. Proceedings of the National Academy of Sciences, 111(32), 11852-11857.

[23] Jones, K. J., Templet, S., Zemoura, K., Kuzniewska, B., Pena, F. X., Hwang, H., ... & Xu, W. (2018). Rapid, experience-dependent translation of neurogranin enables memory encoding. Proceedings of the National Academy of Sciences, 115(25), E5805-E5814.

[24] Eldridge, L. L., Knowlton, B. J., Furmanski, C. S., Bookheimer, S. Y., & Engel, S. A. (2000). Remembering episodes: a selective role for the hippocampus during retrieval. Nature neuroscience, 3(11), 1149-1152.

[25] Shaprine, M. L., & Eichenbaum, H. (2000). Hippocampus is a memory map: synaptic plasticity andmemory encoding by hippocampus neuron. Hippocampus, 9, 365-384.

[26] Paulsen, O., & Moser, E. (1998). A model of hippocampal memory encoding and retrieval:

GABAergic control of synaptic plasticity. Trends Neurosci., 273–278.

[27] Jang, A. I., Nassar, M. R., Dillon, D. G., & Frank, M. J. (2019). Positive reward prediction errors during decision-making strengthen memory encoding. Nature human behaviour, 3(7), 719-732.

[28] Wiest, D. J., Wong, E. H., Minero, L. P., & Pumaccahua, T. T. (2014). Utilizing computerized cognitive training to improve working memory and encoding: Piloting a school-based intervention. Education, 135(2), 264-270

[29] Dale, C. L., Brown, E. G., Herman, A. B., Hinkley, L. B., Subramaniam, K., Fisher, M., ... & Nagarajan, S. S. (2020). Intervention-specific patterns of cortical function plasticity during auditory encoding in people with schizophrenia. Schizophrenia research, 215, 241-249