Determinants of remembrance with depression and anxiety disorders.

Nerisa Banaj*

Department of Clinical and Behavioral Neurology, IRCCS Santa Lucia Foundation, Rome, Italy

Abstract

The authors offer a fresh proposal for the neurobiological mechanisms controlling the memory-related PostTraumatic Stress Disorder (PTSD). The present fear-conditioning model takes into account learning, which underlies some of the core symptoms of PTSD, but it does not take into consideration peritraumatic memory abnormalities or memory recall phenomena, which are also signs of the disorder. According to some theories, the hippocampus is where the core of the traumatic experience is organised, and the emotional challenges restriction of hippocampus activity at high emotional arousal levels is what causes the reduction in conscious recollection for peritraumatic events.

Keywords: Post traumatic, Peritraumatic, Hippocampus.

Introduction

Exposure to severe emotional stress, PTSD is a unique anxiety disease with a high frequency and morbidity. The condition is characterized by three symptom clusters, all of which are caused directly or indirectly by memory functions: Persistent re-experience of the traumatic event; Persistent symptoms of heightened arousal; Persistent avoidance of stimuli connected to the trauma, which may involve amnesia for a significant element of the traumatic event [1]. One of the first acquired psychiatric disorders to be logically diagnosed and eventually treated—i.e., based on a clear pathophysiologic mechanism promises to be PTSD. There are two reasons why PTSD has attained this status. First, the proximal cause can be identified and may be seen from outside. Each of the three symptom clusters listed in the standard diagnostic criteria reflects a dysfunction in the way memory functions for particular experiences that may be visible. Second, understanding of the neurobiology of memory has advanced to a point where it is possible to characterise the neurophysiological mechanisms brought on by extremely adverse emotional stimulus with increasing accuracy. It is well known that intricate interactions between specialised memory systems, mediated by different cerebral networks, occur throughout human learning [2].

Restrictions of the present fear-conditioning model

The neurophysiological processes brought on by extremely unpleasant Based on a preclinical concept incorporating animal fear conditioning, progress has been made in understanding the molecular basis of anxiety disorders over the past ten years. In particular, the results of conditioning trials, especially in mice, serve as an analogy for the current neurobiological model of PTSD. In these studies, an initially neutral stimulus, such as an auditory tone, causes autonomic fear reactions when it is coupled with a profoundly unpleasant experience, typically an electric shock. The junction nuclei, complexes of neurons located in the temporal lobes, have been identified as being at the centre of the neuro anatomic signals responsible for the perception, expression, and memory of fear through studies of fear conditioning using brain lesions, drugs, and electrophysiological methods. Fear conditioning, which is independent of and may not include conscious memory of experience, is the process of learning specific autonomic reactions. It has been shown through experimental research and clinical observations of neurologically damaged people that this type of conditioning can take place even in the absence of conscious recollection for traumatic or unpleasant occurrences. Interestingly, the fear-conditioning model effectively explains the origin of some PTSD symptoms caused by memory strengthening [3].

Depending on a preclinical concept incorporating animal fear conditioning, progress has been made in understanding the molecular basis of anxiety disorders over the past ten years. In particular, the results of conditioning trials, especially in mice, serve as an example for the current neurobiological model of PTSD. In these studies, an initially neutral input, such as an auditory tone, causes autonomic fear reactions when it is combined with a profoundly unpleasant experience, typically an electric shock. The junction nuclei, complexes of neurons located in the temporal lobes, have been identified as being at the centre of the neuroanatomical circuitry responsible for the perception, expression, and memory of fear through studies of fear conditioning using brain lesions, drugs, and electrophysiological methods. The best way to conceptualise PTSD is as a clinical illness that includes memory impairment for the context of the trauma, as well as dissociation of the experience from regular autobiographical memory, as well

***Correspondence to:** Nerisa Banaj, Department of Clinical and Behavioral Neurology, IRCCS Santa Lucia Foundation, Rome, Italy. E-mail: nerisabanaj@hsantalucia.it

Received: 01-*Sep-2022, Manuscript No. AAJNNR-22-75769;* ***Editor assigned:*** *05-Sep-2022, Pre QC No. AAJNNR-22-75769 (PQ);* ***Reviewed:*** *19-Sep-2022, QC No. AAJNNR-22-75769;* ***Revised:*** *21-Sep-2022, Manuscript No. AAJNNR-22-75769 (R);* ***Published:*** *28-Sep-2022, DOI: 10.35841/aajnnr-7.5.122*

Citation: *Banaj N. Determinants of remembrance with depression and anxiety disorders. J Neurol Neurorehab Res. 2022;7(5):122*

as memory intensification for the core traumatic event. It has generally been assumed that these conscious memory disturbances are secondary symptoms that develop as psychological defences to suppress and otherwise avoid the primary symptoms of intensified memory [4].

Consequences for research and treatment

A neurobiological explanation of the cognitive memory symptoms of PTSD is supported by laboratory studies of the connection between memory intensification and amnesia. Additionally, they provide a strategy that would enable the experimental manipulation necessary to test the given neurophysiological process theories. To clarify neurophysiological mechanisms, pharmacological alterations of the von Restorff technique are particularly important since they allow exact adjustment in the parameters of the crucial stimulus. Additionally, the von Restorff paradigm allows for the research of putative neurobiological pathways underlying PTSD in clinical and nonclinical human populations because it is a safe methodology absent of fear conditioning. The involvement of the amygdala and hippocampus in memory enhancement and amnesia may be better understood with the use of selective pharmacological suppression in nonclinical human patients.

Using the von Restorff paradigm to examine the model's hypothesis for the modification of the interaction of the amygdala and hippocampus would be a logical place to start. Functional MRI and selective pharmacological inactivation of the hippocampus or amygdala could be utilized to examine how these regions respond to stimuli of various intensities. Correlational research might then verify or refute hypothesised associations between amygdala activity and changes in memory at different intensities of a crucial stimulus [5].

Conclusion

The medial and orbital prefrontal cortex and the amygdala have strong reciprocal connections, and research has demonstrated that the prefrontal cortex can regulate amygdala activity. Additionally, the hypothesis medial prefrontal cortex can control amygdala function related to emotional learning is supported by specific behavioural effects of prefrontal dysfunction that have been shown in both animals and humans. These findings imply that the prefrontal cortex may be a possible target for therapy intervention and that the integrity of the prefrontal area may be one element that predicts level of vulnerability for development of PTSD. The prevention or extinction of conditioned fear reactions has naturally been the focus of proposed treatments for PTSD based on the fear-conditioning model. In an effort to reduce the conditioning of fear, treatment strategies focused at pharmacologically suppressing amygdala activity close to the time of trauma exposure have shown rare success.

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Citation: *Banaj N. Determinants of remembrance with depression and anxiety disorders. J Neurol Neurorehab Res. 2022;7(5):122*