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Psychological Effects Based on Phenomenological Descriptions

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Abstract

Post-traumatic stress disorder has been linked to the dopamine, norepinephrine, endorphin, serotonin, and oxytocin systems, as well as the mesocortical and mesolimbic dopamine, norepinephrine, endorphin, serotonin, and oxytocin systems. On the other hand, very little is known about how these different systems interact, and no well-known unified theory has yet emerged. This review suggests that galanergic suppression of dopaminergic neurons in the ventral tegmental might be the missing link in a post-traumatic feedback loop.

Keywords: Trauma • Stress disorder • Anxiety • Mesolimbic dopamine

Introduction

The debilitating mental condition known as Post-Traumatic Stress Disorder (PTSD) is characterized by symptoms that are both hypodopaminergic and hyperadrenergic. Other neuroendocrine systems, such as the oxytocin system, serotonin system, and -opioid system, have also been linked to PTSD. NE hyperfunction and DA hypofunction have been linked to the condition. As a result, a conclusive theory has not yet emerged as to whether noradrenergic dysregulation and/or dopaminergic hypofunction are fundamental etiological features of the condition or merely symptoms of a different neurological process. The neuropeptide galanin (Gal), which has received less attention, may play a significant role in a post-traumatic positive feedback loop, according to our hypothesis in this study. So, unique genetic predispositions and environmental stressors like the type of trauma, social connection after traumatization, and coping strategies may play a significant role in the development of PTSD. Second, the four main categories of neurotropic PTSD treatments are as follows: medications that block NE binding (such as 1- and -adrenergic receptor antagonists), medications that bypass the VTA and directly stimulate mesocorticolimbic DA receptors in the mPFC and Nacc, and atypical antipsychotics are examples of medications that reduce LC reactivity. Gal is co-secreted with NE by about 80% of locus coeruleus (LC) neurons (VTA) and inhibits dopaminergic projections from the ventral tegmental region [1].

Literature Review

The evidence suggests that Gal has a significant impact on DA projections to the nucleus accumbens (Nacc) and the medial prefrontal cortex (mPFC), although it is unclear whether Gal affects all VTA neurons or just a subset of them. DA abnormalities in these pathways have been linked to PTSD's hypodopaminergic symptoms and faulty fear-extinction processes. The -opioid receptor (MOR) in the VTA interacts cross-antagonistically with the Gal Receptor 1 (GalR1). With the exception of atypical antipsychotics, none of these medications have yet demonstrated long-term efficacy in large population samples, despite the fact that they all imply a direct or indirect downregulation or interruption of this feedback loop. However, there is no guarantee that a disruption will have long-term effects because this feedback loop is not a closed system. Subsequently, MOR feeling, especially through conduct and social mediation, may open up new roads in the treatment of PTSD, with huge ramifications for enrollment, preparing, and authority processes in high-stress/high-risk callings like the military, people on call, and police [2].

Discussion

A cluster of cases of mucormycosis were identified in a 6-year retrospective chart review of 16 Texas patients during the months of February and March, when the average temperature rarely exceeded 25°C. The invasive fungal infections' demographics are similar to those of the general trauma population, with a male predominance and a mean age of 27 to 48. A 23-year-old male suffered a rare fatal case of mucormycosis tracheitis following blunt trauma in a West Texas woodland. Gal is a neuropeptide that is found in a lot of places in the central nervous system of mammals. About 80% of NE neurons in the LC express it. LC-derived Gal innervates the cortex, hypothalamus, hippocampus, and VTA all at the same time. Additionally, cross-antagonistic interactions between GalR1 and MOR in functional heteromers have been found in recent research. MOR are typically found in the tail of the VTA, which consists primarily of GABAergic neurons and controls VTA DA projections, despite the possibility of a wider distribution. This suggests that GalR1 is abundant in this region. Consequently, the VTA appears to express both GalR1 and GalR2; However, the VTA may have different distributions of the two types of receptors [3].

The majority of studies focus on galaninergic modulation of mesolimbic DA rather than the behavior-affecting effects of LC-derived Gal. Anhedonia, decreased proactive coping, and a slowed rate of recovery following a stressful event are all attributed, at least in part, to galanergic inhibition of DA neurons in the VTA, according to the current evidence from a variety of rat models. However, these studies use phenomenological descriptions of behavioral patterns to infer psychological effects, which may or may not be predictive of the implied effects. Consequently, additional research is required before precise conclusions can be drawn. The LC receives glutamate from the paragigantocellularis nucleus, lateral habenula, and mPFC, the predominant excitatory input in this region of the brain. This last input is most likely controlled by the D2R, which explains the role of mPFC DA in fear extinction and suggests a feedback loop in which enhanced glutaminergic activation of the LC in PTSD is made possible by suppressing mesocorticolimbic DA by galanergic activity during LC hyperfunction. Hyperadrenergic and hypodopaminergic problem clusters in PTSD may be explained by this LC-VTA-mPFC/Nacc-LC feedback loop; consequently, a disruption should be predicted to alleviate symptoms that are directly and indirectly related to the disruption [4].

The LC receives glutamate from the paragigantocellularis nucleus, lateral habenula, and mPFC, the primary excitatory input in this brain region. This latter input is probably controlled by the D2R, which explains the role of mPFC DA in fear extinction and suggests a feedback loop in PTSD in which higher glutaminergic stimulation of the LC is supported by the suppression of mesocorticolimbic

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DA by galanergic activity during LC hyperfunction. Hyperadrenergic and hypodopaminergic symptom clusters in PTSD may be explained by this LC-VTA-mPFC/Nacc-LC feedback loop, and a disruption should be expected to alleviate symptoms that are directly related to the disturbance as well as downstream processes. The LC-VTA-mPFC/Nacc-LC feedback loop is affected directly or indirectly by each of these systems, and their distribution may vary depending on the patient population. Because of this, it is highly unlikely that any of them will be found to be significant in a large Genome-Wide Association study but not in smaller subpopulation analyses [5].

Conclusion

Galanin might assume a significant part in PTSD, as per one survey. Due to poor regulation of glutaminergic projections from the mPFC to the LC, galaninergic suppression of mesocorticolimbic dopamine has been linked to both hypodopaminergic and hyperadrenergic symptom clusters in PTSD patients. However, this feedback loop is not a closed system, which highlights the need for a more holistic approach to PTSD rehabilitation and may explain why many neurotropic medications fail to have a long-term effect. Additionally, we think that GalR1-MOR heteromers could be a target for new treatments and preventative measures.

Acknowledgement

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Conflict of Interest

None.

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