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Post-traumatic Stress Disorder (PTSD) is a Type of Anxiety Illness that Occurs

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Abstract

The dopamine, norepinephrine, -endorphin, serotonin, and oxytocin systems, as well as the mesocortical and mesolimbic dopamine, norepinephrine, -endorphin, serotonin, and oxytocin systems, have all been linked to post-traumatic stress disorder. The interaction between these various systems, on the other hand, is largely unknown, and a widely recognised unified theory has yet to emerge. Galanergic suppression of dopaminergic neurons in the ventral tegmental may be the missing link in a post-traumatic feedback loop, according to this review.

Keywords: Post-traumatic stress disorder • Anxiety

Introduction

Post-traumatic Stress Disorder (PTSD) is a debilitating mental condition characterised by both hyperadrenergic and hypodopaminergic symptoms. Although Norepinephrine (NE) hyperfunction and Dopamine (DA) hypofunction have been linked to PTSD, various other neuroendocrine systems, such as the oxytocin system, serotonin system, and -opioid system, have also been linked to the illness. As a result, it's unclear if noradrenergic dysregulation and/or dopaminergic hypofunction are a basic etiological aspect of the condition or only a symptom of another neurological process and a unifying hypothesis has yet to emerge. In this study, we propose that the neuropeptide galanin (Gal), which has received less attention, may play a vital role in a post-traumatic positive feedback loop. Gal inhibits dopaminergic projections from the ventral tegmental region and is co-secreted with NE by about 80% of locus coeruleus (LC) neurons (VTA) [1,2].

Although it is unclear whether Gal affects all VTA neurons or just a subset of them, the evidence suggests that it has a significant impact on DA projections to the nucleus accumbens (Nacc) and the medial prefrontal cortex (mPFC). The hypodopaminergic symptoms seen in PTSD, as well as faulty fear-extinction processes, have been linked to DA abnormalities in these pathways. The Gal Receptor 1 (GalR1) has cross-antagonistic interactions with the -opioid receptor (MOR) in the VTA. As a result, MOR stimulation, particularly through behavioural and social intervention, may open up new avenues in the treatment of PTSD, with significant implications for recruitment, training, and leadership processes in high-stress/high-risk professions like the military, first responders, and cops [3-5].

In a 6-year retrospective chart review of 16 patients in Texas, a cluster of mucormycosis cases were noted during the months of February and March when the average temperature rarely exceeded 25ºC [6]. The demographics of invasive fungal infections reflect that of the general trauma population with a male predominance and a mean age between 27 and 48 years old [7]. Here, we report a rare fatal case of mucormycosis tracheitis in a 23-year-old male after blunt trauma in a woodland area in West Texas.

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Galanin with the Stress Response

Gal is a widespread neuropeptide found throughout the mammalian central nervous system, with around 80% of NE neurons in the LC expressing it. The cortex, hypothalamus, hippocampus, and VTA are all innervated by LC-derived Gal. Furthermore, some recent studies reveal that GalR1 interacts with MOR in functional heteromers in a cross-antagonistic manner. Although a wider distribution is feasible, MOR are typically found in the tail of the VTA, which comprises largely GABAergic neurons and controls VTA DA projections, implying a strong presence of GalR1 in this region. As a result, it appears that the VTA expresses both GalR1 and GalR2; however the two receptor types may be distributed differently across the VTA [6].

The effects of LC-derived Gal on behaviour are uncommon, and most investigations focus on galaninergic modulation of mesolimbic DA. The present evidence, obtained from a variety of rat models, suggests that galanergic inhibition of DA neurons in the VTA, at least in part, causes anhedonia, reduces proactive coping, and slows recovery after a stressful event. These studies, however, infer psychological effects based on phenomenological descriptions of behavioural patterns, which may or may not be predictive of the implied effects. As a result, more research is required before any clear conclusions can be formed.

Post-traumatic Feedback-loop

The paragigantocellularis nucleus, the lateral habenula, and the mPFC all send glutamate to the LC, which is the predominant excitatory input in this brain region. This latter 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 the suppression of mesocorticolimbic DA by galanergic activity during LC hyperfunction facilitates enhanced glutaminergic activation of the LC in PTSD. This LC-VTA-mPFC/Nacc-LC feedback loop could explain both hyperadrenergic and hypodopaminergic problem clusters in PTSD, and a disruption should thus be predicted to ameliorate symptoms that are both directly and indirectly related with the disruption [7].

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These systems all have an effect on the LC-VTA-mPFC/Nacc-LC feedback loop, either directly or indirectly, and may be distributed differentially across different patient populations. As a result, it's unlikely that any of them will emerge as important in a big Genome-Wide Association research but not in smaller sub-population analyses. As a result, the complex interplay between unique genetic predispositions and environmental stressors such as trauma type, social connection in the aftermath of traumatization, and coping methods may play a substantial role in the development of PTSD. Second, most neurotropic treatments for PTSD fall into one of four categories: medications that reduce LC reactivity (e.g., 2-adrenergic receptor agonists and 5-HT agonists), medications that block NE binding (e.g., 1- and -adrenergic receptor antagonists), medications that bypass the VTA and directly stimulate mesocorticolimbic DA receptors in the mPFC and Nacc, and atypical antipsychotics. Despite the fact that, with the exception of atypical antipsychotics, all of these drugs imply a direct or indirect down-regulation or interruption of this feedback loop, none has yet demonstrated long-term efficacy in large population samples. However, because this feedback loop isn't a closed system, a disruption isn't guaranteed to have long-term consequences.

Conclusion

Galanin may play a major role in PTSD, according to one review. Galaninergic suppression of mesocorticolimbic dopamine has been linked to both hypodopaminergic and hyperadrenergic symptom clusters in PTSD patients, owing to poor regulation of glutaminergic projections from the mPFC to the LC. However, this feedback loop is not a closed system, which may explain why many neurotropic drugs fail to have a long-term effect and highlights the need for a more holistic approach to PTSD rehabilitation. We also believe that GalR1-MOR heteromers could be used as a target for new therapy and preventive methods.

Conflict of Interest

No conflict of interest by author.

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