



Brief Review on the Cortico-Striato-Thalamo-Cortical Loops and their Roles in Anxiety and Worry

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Abstract

Anxiety is a common pathology in the modern world. Understanding the basic mechanisms involved in the occurrence of such pathology is of great importance to treat the patients suffering from such pathology better. This brief review tries to point to few of such mechanisms.

Keywords: Cortico-striato-thalamo-cortical loops; Anxiety; Worry

Body

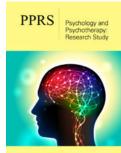
The Cortico-Striato-Thalamo-Cortical loops are circuits of the brain which control execution of movement, formation of habit and reward. "Worry" which would be the second core symptom of anxiety disorders is relevant to the prefrontal cortex Cortico-striato-thalamo-cortical feedback loops [1]. Catastrophic thinking, apprehensive expectations, anxious misery and obsessions can be seen in "worry" in anxiety disorders. The Cortico-striato-thalamo-cortical feedback loops may be involved in delusions, ruminations and obsessions either. Such circuits can be modulated by various regulators and neurotransmitters like dopamine, serotonin, glutamate, GABA, norepinephrine and together with voltage-gated ion channels modulate the amygdale. Amygdala-centered circuits dysfunction is linked to the appearance of anxiety symptoms [2]. Overactivation of the Cortico-striato-thalamo-cortical loop which originates and ends in the Dorsolateral prefrontal cortex may result in the appearance of worry symptoms. The same can occur with the appearance of obsessions symptoms [3].

Understanding these mechanisms is important to treat anxiety, its symptoms and relevant pathologies. As a few examples, amygdala circuits overactivation can lead to the appearance of pathological anxiety or fear. Benzodiazepines like other GABAergic agents, by phasic inhibitory actions enhancement at the amygdala postsynaptic GABA a receptors, can decrease the appearance of anxiety or fear symptoms [4-6]. Serotonergic neurons would send some signals to the amygdala which can inhibit some of its outputs. By increasing the amounts of such signals with using serotonergic agents, it can be possible to alleviate anxiety or fear. Increased amounts of glutamate release in the amygdale can lead to the appearance of anxiety symptoms. Blocking such increased release by using certain agents which can bind to the certain subunit of the N and P/Q voltage-sensitive calcium channels at the presynaptic point, can also decrease the amounts of anxiety symptoms [7].

Conclusion

Understanding the basic neurobiological mechanisms which are involved in the appearance of anxiety or fear symptoms is of great importance to control such symptoms at the bedside much more effectively. Also such understanding would lead to finding possible novel treatment agents which can be used in the patients suffering from such pathologies.

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