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MEETING ABSTRACTS

ON THE ROLE OF OPIOID-GALANIN RECEPTOR HETEROMER FUNCTION IN POST-TRAUMATIC STRESS DISORDER: A POTENTIAL TARGET FOR TREATMENT OR A NEUROENDOCRINOLOGICAL DEAD-END?

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There are strong indications that the μ-Opioid system plays a role in the aetiology of post-traumatic stress disorder (PTSD). Exogenous stimulation of the μ-Opioid receptor (MOR) with opioid medication in the wake of a traumatic experience attenuates sensitivity to PTSD, opiate addiction is a common comorbid disorder with PTSD, and there are indications that engaging in behaviours which stimulate endogenous β-endorphin, a natural MOR ligand, outflow can suppress symptoms in those who have the disorder. The latter is especially relevant with regard to post-combat PTSD, because many elements of military life stimulate social synchronisation, which has been linked to enhanced β-endorphin function. Recently, however, several independent studies have identified cross-antagonistic interactions between the MOR and Galanin 1 Receptor (GalR1) within functional heteromers in the tail of the Ventral Tegmental Area. This interplay between MOR and GalR1 may explain some of the specific symptom clusters observed in post-combat PTSD and could, potentially, open the doors to new treatment options. In this paper, I will address the literature on MOR-GalR1 interactions and assess the extent to which the available evidence supports further exploration of this pathway in the search for more effective treatments of combat-related PTSD.

Keywords: post-traumatic stress disorder; MOR-GalR1 heteromers; β-endorphin

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