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Cellular and Molecular Adaptations From Cue-Induced Cocaine Relapse Within VTA-Projecting Ventral Pallidum Neurons in Mice

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Cocaine induces adaptations and alterations within the reward circuitry that promote cocaine-seeking. The ventral pallidum (VP) is one node within the reward pathway known to receive input from and projects to several regions that mediate cocaine-seeking. This includes VP afferents to the ventral tegmental area (VTA-projecting VP) which are required for cocaine relapse. However, the cellular and molecular adaptations that occur within VTA-projecting VP neurons to promote cocaine-seeking remain poorly understood. In the present study, we demonstrate that selective inhibition of VTA-projecting VP neurons effectively reduces cue-induced cocaine reinstatement in both male and female mice following intravenous cocaine-self administration (IVSA). Additionally, we observe a significant increase in cFos expression in the VP following cue-induced reinstatement. Ongoing investigations are focused on identifying specific VP cell types activated during reinstatement. Moreover, we are exploring the impact of cocaine IVSA on dendritic and spine morphology within VTA-projecting VP neurons. Collectively, these studies aim to elucidate the neural adaptations within the VP circuitry that underpin cocaine relapse.