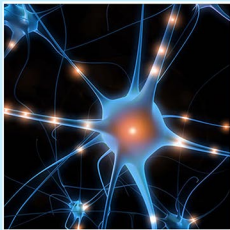




SHAY DAJI

Faculty Research Mentor: Dr. Todd Thiele
Department of Psychology



Neurons Firing Action Potentials

(Image credit: http://www.spring.org.uk/images/brain_cell.jpg)

Binge Alcohol Consumption Behavior Does Not Induce IL-10 Immune Response in the Ventral Tegmental Area of the Mouse Brain

Cytokines, such as the anti-inflammatory Interleukin-10 (IL-10), are major mediators of the neuroimmune response to binge levels of alcohol and have been shown to play a role in controlling inflammation. Pro-inflammatory signaling has been linked with high alcohol preference and other behavioral changes in both mice and humans. Drug-induced activation of pro-inflammatory central immune signaling has been shown to contribute to abusive behavior specifically by enhancing the engagement of classical mesolimbic dopamine reward pathway. IL-10 has been implicated in maintaining Th1/Th2 immune cytokine balance and may play a role in preventing excess inflammation in this pathway. Administration of IL-10 prior to induced proinflammatory signaling has been shown to revert binge alcohol consumption behavior, however the effect of binge alcohol consumption on IL-10 immunoreactivity in the mesolimbic pathway has yet to be investigated. In this study, the effect of binge alcohol consumption on IL-10 immunoreactivity within the Ventral Tegmental Area (VTA) of the mesolimbic pathway was assessed. We followed the "Drinking in the Dark" (DID) paradigm, a model of binge-like ethanol drinking in C57BL/6J mice. Upon completion of the DID procedure, immunohistochemical staining was used to assess the concentrations of IL-10 in the VTA. These results suggest that the lack of a neuroimmune anti-inflammatory response in the VTA could be a possible factor intensifying the inflammation of the mesolimbic reward pathway that underlies the behavioral maladaptations that lead to alcoholism.