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DRINKING HISTORY ENHANCES DOPAMINE NEURONAL RESPONSE TO ETHANOL

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Ethanol (EtOH) increases the activity of dopamine (DA) neurons in the ventral tegmental area (VTA) in vivo and in vitro, although relatively high doses of acute EtOH are often required to cause a significant increase of the firing rate of VTA DA neurons in vitro. In order to determine whether prior exposure to ethanol results in an increase in the sensitivity of VTA DA neurons to the drug, the firing of neurons from alcohol-naïve mice was compared to the firing of neurons from mice which had undergone repeated ethanol consumption.

We have used an intermittent access protocol to produce escalated voluntary alcohol intake and preference for alcohol over water in male mice. The results from this experiment suggest that neurons from mice which have learned to drink during adolescence demonstrate an increased sensitivity to alcohol compared to neurons from naive individuals. The baseline spontaneous firing rate and resting membrane potential of VTA DA neurons do not significantly differ between the two groups of mice, suggesting that alcohol experience does not alter intrinsic excitability of these neurons. This alteration in sensitivity is a unique property of A10 (VTA) DA neurons, as DA neurons in the neighboring A9 (SN) region show no significant effects after alcohol consumption. However, we do observe evidence for regional heterogeneity within the VTA, as the effects of drinking history are more pronounced in medial VTA DA neurons, compared to lateral VTA. These results will be helpful in elucidating the neuronal adaptations that occur after repeated alcohol use.