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Acute Ethanol Modulation of Neurocircuit Function in the Nucleus of the Tractus Solitarius

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Highlights

- EtOH enhances pre- and post-synaptic GABAergic transmission in the NTS
- EtOH has minimal effects on NTS glutamatergic transmission
- EtOH decreases action potential firing in a majority of neurons
- EtOH enhances c-FOS/tyrosine hydroxylase colocalization in the NTS
- EtOH may disinhibit NTS TH neurons by increasing GABA signals between interneurons

Abstract

The nucleus of the tractus solitarius (NTS) is a brain stem region critical to many physiologic processes and has been implicated in addiction to multiple classes of abused drugs, including alcohol (EtOH). That said, the mechanism by which EtOH modulates NTS neurocircuit activity is not well characterized and has yet to be examined utilizing electrophysiologic methods in mouse models of alcohol use disorders. To begin to address this gap in knowledge, we sought to use whole-cell and cell-attached recordings to determine the mechanism of acute EtOH action on GABAergic and glutamatergic neurotransmission, as well as on action potential firing in the NTS of adult male, EtOH naïve mice. Bath application of EtOH (50 mM) significantly enhanced the frequency of spontaneous inhibitory postsynaptic current events, while increasing the amplitude of these events in half of the neurons tested. This finding suggests a presynaptic mechanism of EtOH action on GABAergic transmission in the NTS as well as a postsynaptic mechanism in subsets of NTS neurons. EtOH application was further associated with a significant decrease in action potential firing in most, but not all, NTS neurons tested. EtOH induced a small but significant decrease in spontaneous excitatory postsynaptic current frequency,