Neuroscience 2013

Print this Page for Your Records Close Window

Control/Tracking Number: 2013-S-5999-SfN
Activity: Scientific Abstract
Current Date/Time: 5/7/2013 4:24:08 PM

# The effect of noise trauma on Arc and GAD expression in a rat model of tinnitus

AUTHOR BLOCK: *M. R. KAPOLOWICZ, E. H. KARDOSI, F. K. ALAHMADY, J. I. SEDILLO, L. T. THOMPSON; Behavioral and Brain Sci., The Univ. of Texas at Dallas, Richardson, TX

*Abstract:*
Tinnitus is an auditory hallucination of sound, a devastating condition that is affecting a growing number of people each year. Treating patients who suffer from chronic tinnitus has proven difficult due to an inability to characterize the abnormal plastic changes that initiate and maintain tinnitus. The aim of this experiment was to better understand the brain mechanisms involved in the initial plasticity that may be contributing to the emergence of this disorder. Specifically, the focus was to address amygdalo-hippocampal involvement in the early stages of tinnitus. We characterized changes in both excitatory and inhibitory signaling in these regions after acute noise trauma, which is the most common cause of tinnitus in humans. To assess excitatory involvement of these limbic regions, Arc protein expression was evaluated in male rats ($n$ = 24) shortly after being bilaterally exposed to acute high-intensity noise (16 kHz, 115 dB, for 1 hr) that has been proven effective in causing acute cochlear trauma and development of behavioral signs of tinnitus in rats. Western blot analysis confirmed that amygdalo-hippocampal Arc expression was up-regulated in some regions yet down-regulated in others post-noise trauma. Western blot analysis also revealed regionally-specific changes in protein expression of GAD, the biosynthetic enzyme required for GABAergic inhibition, as a result of noise trauma. This corroborates other evidence indicating that these limbic structures which are located outside of the classical auditory pathway may be involved in the manifestation of tinnitus.
: