(632) Chronic vibration reduces thresholds of A-beta and A-delta nerve fibers in the rat tail

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Exposure to hand-arm vibration through the use of power hand-tools can result in the development of hand-arm vibration syndrome (HAVS), which is characterized by a reduction in tactile sensitivity in the fingers and hands of workers. The etiology underlying the development of HAVS is unknown. The goal of this study was to use a rat-tail model of HAVS to characterize progressive changes in sensory nerve function that occur with repeated exposures to vibration. Rats were exposed to tail vibration, restraint or control conditions 4 h/day, 5 days/week for 5 weeks. A-beta, A-delta and C fiber function was assessed using transcutaneous electrical stimulation at 3 different frequencies. Thresholds were measured before and after the last exposure each week. Analyses revealed that between week 1 and week 2, A-beta and A-delta fiber thresholds declined by about 50% in vibrated rats, indicating an increased sensitivity to mechanical and pain stimuli. Vibration also caused an additional transient reduction in A-beta fiber thresholds immediately following exposures during weeks 4-5. No changes were seen in C-fiber thresholds over time. Although HAVS is associated with a reduction in tactile sensitivity, this loss of sensory function may be preceded by hyperalgesia to mechanical stimuli. Thus, this increase in pain sensitivity may serve as an early marker of vibration-induced nerve damage, and non-invasive tests to detect mechanical hyperalgesia could be used to identify workers that may be at risk for developing HAVERSUS. (The findings and conclusions in this abstract have not been officially disseminated by NIOSH and should not be construed to represent any agency determination or policy.)

(634) Microinjection of NMDA and non-NMDA antagonists into the central nucleus of the amygdala suppresses pain affect in rats

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Society for Neuroscience abstract

B04 - Brainstem, Thalamus, & Cortex

(633) Alterations in forebrain activation patterns associated with neuropathic pain in Zucker type 2 diabetic rats

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A significant percentage of patients with type 2 diabetes mellitus develop peripheral polyneuropathy and experience severe and unremitting chronic neuropathic pain. This condition is among the most difficult to manage and often resists effective treatment altogether. This pain can be debilitating, frequently leads to physical impairments and functional limitations and can significantly impact on the quality of life. The pathophysiology of the diabetic neuropathy and its painful variant remain poorly understood. The Zucker diabetic fatty (ZDF) rat is an animal model of Type 2 diabetes, characterized by the spontaneous onset of diabetes at approximately 70 days of age. This model shows insulin resistance with hyperglycaemia and hyperinsulinea and as such closely mimics human Type 2 diabetes. Increasing our knowledge of the CNS mechanisms involved in painful Type 2 diabetic neuropathy should facilitate the development of treatments that are more effective and specific than those currently available. Accordingly, we tested ZDF and Zucker lean non-diabetic control rats for the presence of neuropathic pain by measuring behavioral responses to innocuous mechanical and noxious thermal stimuli. Diabetic rats showed increased sensitivity to stimuli by 10 weeks of age compared to the lean control rats. This hyperresponsiveness lasted for up to 14 weeks of age, at which time we evaluated the changes in brain activation associated with diabetes-induced neuropathic pain. We found differences in the basal (unstimulated) activation patterns in diabetic rats with neuropathic pain compared to age matched non-diabetic control animals in several supraspinal regions. For example, compared to control rats, ZDF rats showed significant increases in activation in somatosensory and limbic structures. Our results suggest that, as a consequence of and in parallel to the development of a diabetic neuropathy, maladaptive alterations occur in the functional activation of multiple CNS structures involved in pain perception and/or pain modulation.

(635) Regional differences within the anterior cingulate cortex in antinociception elicited by NMDA agonists and antagonists

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