Neuropeptide Y Modulates c-Fos Protein Expression in the Cuneate

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摘要

Abstract

This study sought to investigate the effects of injury-induced neuropeptide Y (NPY) on c-Fos expression in the cuneate neurons and neuropathic pain after median nerve injury. Four weeks after median nerve transection (MNT), the injured nerves stimulated at low intensity (0.1 mA) expressed significantly less NPY-like immunoreactive (NPY-LI) fibers in the cuneate nucleus (CN) than those stimulated at high intensities (1.0 mA and 10 mA). Conversely, a significantly higher number of c-Fos-LI cells were observed in the CN in rats stimulated with 0.1 mA compared to those stimulated with 1.0 mA or 10 mA. These results suggest that more NPY was released following low-intensity stimulation, and consequently fewer NPY-LI fibers and more c-Fos-LI cells were identified in the CN. Furthermore, the number of c-Fos-LI cells as well as the percentage of c-Fos-LI cuneothalamic projection neurons (CTNs) in the CN was markedly decreased after injection of NPY receptor antagonist along with retrograde tract-tracing method, indicating that NPY regulated c-Fos expression. In rats with median nerve chronic constriction injury (CCI), intracerebroventricular injection of NPY aggravated mechanical allodynia and low-intensity stimulus-evoked c-Fos expression, both of which were reversed by injection of NPY receptor antagonist. However, thermal hyperalgesia was not affected by injection of these two reagents. Taken together, these findings suggest that more NPY release, following low-intensity electrical stimulation of the injured nerve, significantly induces c-Fos expression in the CTNs, which possibly provide the ascending thalamic transmission of neuropathic pain signals.