The role of cell adhesion molecules and neurotrophic factors in reconstruction of nociceptive pathways in spinal cord and brainstem after peripheralaxotomy

2000 Fiscal Year Final Research Report Summary

The role of cell adhesion molecules and neurotrophic factors in reconstruction of nociceptive pathways in spinal cord and brainstem after peripheralaxotomy

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Cerebral neurosurgery

Research Institution

Kanazawa University

Principal Investigator

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Research Abstract

The ultimate aim of this study is to know the plasticity of primary sensory pathways, especially pain sensation in the spinal cord, resulting in allodynia, hyperalgesia, and persistent pain to the patients. To examine this, the effects of peripheral axotomy to the alteration of the expression of E-cadherin which is exclusively expressed inlamina II of Rexed in the spinal cord dorsal horn was firstly analysed. This expression tem porarily disappeared by day 7 after axotomy and reappeared following partial axonal regeneration on day 63. In contrast, it remained undetectable following complete nerve degeneration. Cadherin-associated protein, catenins are also examined. Administration of NGF rescued the immunoreactivity of substance P, which is known to disappear after peripheral axotomy, but not influence that of both E-cadherin and alpha N-catenin. Secondly, to investigate the detailed cellular effects of oxidized galectin-1, which effect to nerve regeneration has been recently identified, acellular auto- and allograft model were utilized. Our results indicated that local application of exogenous rhGal-1/Ox promotes the migration of Schwann cells followed by axonal regeneration from both motor and sensory neurons, and that Gal-1/Ox is a key factor of initial stage of neuronal regeneration. These models would be utilized for further investigation of the plasticity of primary sensory pathways.

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