The role of cell adhesion molecules in reconstruction of neural network in spinal cord and spinal nerves with special attention paid to E-cadherin and its associated protein catenin

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	作成者: Hasegawa, Mitsuhiro
	メールアドレス:
	所属:
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The role of cell adhesion molecules in reconstruction of neural network in spinal cord and spinal nerves with special attention paid to E-cadherin and its associated protein catenin

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Cerebral neurosurgery
Research Institution
Kanazawa University
Principal Investigator
HASEGAWA Mitsuhiro Kanazawa University Department of Neurosurgery Assistant Professor, 医学部附属病院, 講師 (70218460)
Co-Investigator(Kenkyū-buntansha)
TAKEICHI Masatoshi Kyoto University Department of Biophysics Faculty of Science Professor, 理学部, 教授 (00025454)
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E-cadherin / alpha N-catenin / Schwann cell contact / cell adhesion molecule / regeneration / remyelination / spinal cord dorsal horn / axonal sprouting

Research Abstract

An increasing number of cell adhesive proteins is being identified as involved in cell-cell and cell-extracellular matrix interactions. Extensive studies have focused on several members of molecules including L1, N-CAM, MAG during the development and regeneration of the nervous system. We have recently found that Ca2+-dependent cell adhesion molecule E-cadherin plays an important role in the selective fasciculation of a particular subset of unmyelinated sensory fibers and that E-cadherin is exclusively expressed in lamina II of Rexed in the spinal cord dorsal horn. In this study, the temporal profile of the cellular and subcellular expression of E-cadherin and cadherin-associated protein alpha N-catenin in both spinal nerves and lamina II of the spinal cord was examined after crushing, transecting or ligaturing the sciatic nerve in mice. Peripheral nerve injury results in histological and histochemical changes in neurons and glia. Schwann cells proliferated and migrated to form Schwann cell columns (Bungner's bands) as initial responses to denervation, and expressed E-cadherin at their site of contact with each other and later with sprouting axons. At the initial stage of myelin formation, slender processes of a single Schwann cell interdigitated with and enveloped axons, and expressed E-cadherin at the contact site elaborated by a single Schwann cell. The expression of E-cadherin in lamina II disappeared by day 7 after axotomy and reappeared following nerve ligature (partial axonal regeneration model) on day 63. In contrast, it remained at the reduced level after nerve clipping (parmanent axonal degeneration model). The alteration of alpha N-catenin immunoreactivity showed the similar pattern. On the basis of present data, it is suggested that E-cadherin might be involved in the stabilization of peripheral glial network which provides the guidance of sprouting axons and myelination, and that E-cadherin-alpha N-catenin complex might be crucial for plasticity of the spinal cord dorsal horn after peripheral axotomy. Less

Research Products (2 results)

All Other All Publications (2 results)

[Publications] M. Hasegawa,: "Localization of E-cadherin in peripheral glia after nerve injury and repair" J Neuropathol Exp Neurol. 55 (4). 424-434 (1996)

[Publications] M.Hasegawa, A.Seto, N.Uchiyama, S.Kida, T.Yamashima, J.Yamashita: "Localization of E-cadherin in peripheral glia after nerve injury and repair" J Neuropathol Exp Neurol. 55(4). 424-434 (1996)

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