Molecular mechanism of regeneration and plasticity of the injured central nervous system

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【Outline of survey】

In the adult mammalian central nervous system (CNS), it is well known that injured axons exhibit very limited regeneration ability. Due to this lack of appropriate axonal regeneration, a traumatic damage to the adult brain and spinal cord frequently causes permanent neuronal deficits such as paralysis. Several axon growth inhibitors in the CNS have been identified in the myelin. These proteins contribute to the lack of regeneration of the injured CNS. However, it is noted that spontaneous functional recovery sometimes occurs following CNS injury. Synaptic plasticity in pre-existing pathways and the formation of new circuits through collateral sprouting of lesioned and unlesioned fibers are important components of this spontaneous recovery process. The molecular mechanism of this phenomenon is poorly understood, and elucidation of the mechanism will contribute to enhancement of functional recovery after injury to the CNS. Our project will focus on regeneration and plasticity of destructed neural network in the CNS.

【Expected results】

This project will unveil the mechanism of plasticity of neural circuit after injury to the adult CNS, although it had been believed that adult CNS does not regenerate if damaged. Our research is expected to establish a new field of neuroscience. Our goal is to develop therapeutic strategies to enhance intrinsic restoration mechanism. In addition, it may make scientific basis for rehabilitation.

【References】

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【Term of project】 FY2007 － 2011	【Budget allocation】 17,500,000 yen (2007 direct cost)

【Homepage address】 http://www.m.chiba-u.ac.jp/class/neurobio/