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Electrophysiological and Histological Study of Lysolecithin-Induced Local Demyelination in Adult Mice Optic Chiasm

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Abstract

Introduction: Demyelination is one of the main causes of neurological disability. It is the end product of numerous pathological processes, multiple sclerosis (MS) being the most common cause. More than 70% of the MS patients suffer from optic disturbances. This disease commonly affects the optic pathway, particularly the optic nerves and chiasm. Several attempts have been made to produce a suitable model of demyelination in optic apparatus up to now.

Methods: Local demyelination model was generated using direct injection of lysolecithin (LPC) into the optic chiasm of C57/BLJ6 mice without any undesirable distributions of gliotoxin into other brain structures. Histological and electrophysiological assessments of the processes of demyelination and remyelination in the animal model were done with specific myelin staining and visual evoked potential (VEP) recordings.

Results: In this study, both electrophysiological and histological results demonstrated that maximum level of demyelination was observed on day 7 post lesion and an incomplete yet significant remyelination took place on day 14 post lesion.

Conclusion: Results showed a relatively rapid endogenous myelin repair in mice optic chiasm. Furthermore, this report might offer a new tool to address possible involvement of new origins of myelin-forming cells and subsequently their manipulation to promote myelin repair in the adult central nervous system.

Key words: Lysolecithin, Demyelination, Myelin repair, Optic chiasm, Visual evoked potential (VEP), Mouse

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