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少突胶质细胞前体细胞在双环己酮草酰二脲诱导的变化特点(PDF)

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Title: Characteristics of oligodendrocyte precursor cells in cuprizone-induced demyelination models

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关键词: 双环己酮草酰二脲; 脱髓鞘; 髓鞘再生; 少突胶质细胞前体细胞; Olig2; 星形胶质细胞

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摘要: 目的 观察神经胶质细胞在双环己酮草酰二脲（cuprizone，CPZ）诱导的脱髓鞘模型中的变化特点，探讨其与髓鞘再生的内在联系。 方法 选8周龄C57BL/6雄性小鼠，分为正常组、急性脱髓鞘组及髓鞘再生组。正常组每天饲养正常鼠粮；模型组小鼠饲养含有0.2% CPZ的混合鼠粮，连续饲养6周；髓鞘再生组在连续喂养6周CPZ鼠粮后，换用正常鼠粮2周。实验过程中通过卢卡斯快蓝染色及透射电镜技术判断脑组织胼胝体区髓鞘脱失及恢复程度，通过免疫组化及免疫荧光技术检测脑内NG2，Olig2和GFAP的表达来评估少突胶质细胞前体细胞及星形胶质细胞的变化特点。 结果 与正常组相比，急性脱髓鞘组胼胝体髓鞘脱失明显，Olig2、GFAP表达明显升高(P<0.05)，侧脑室旁外侧隔核（LSD）NG2升高水平较胼胝体区（CC）更为明显(P<0.05，P<0.01)；髓鞘再生组髓鞘有所修复，Olig2、GFAP表达仍高于正常组，但较急性脱髓鞘组有一定程度降低(P<0.05)。 结论 髓鞘损伤期少突胶质细胞前体细胞不能有效迁移至受损部位，修复期Olig2表达降低，星形胶质细胞持续高水平表达可能是髓鞘修复障碍的影响因素。

Abstract: Objective To explore the characteristics of oligodendrocyte precursor cells in cuprizone (CPZ)-induced demyelination models. Methods Eight-week old male C57BL/6 mice were randomly divided into a control group, a demyelination group (CPZ 6 weeks) and a remyelination group (CPZ 6 weeks +

normal diet 2 weeks). The mice of the control group were fed with normal diet, and those of the demyelination group and remyelination group were fed with mix diet containing 0.2% CPZ for 6 weeks. Then the mice of the remyelination group were given normal diet instead of CPZ for another 2 weeks. Luxol fast blue (LFB) staining and transmission electron microscopy were used to test the myelin sheath of corpus callosum (CC) in the brain. The expression levels of neuron-glial antigen 2 (NG2), oligodendrocyte transcription factor 2 (Olig2) and glial fibrillary acidic protein (GFAP) in brain tissues were detected by immunohistochemistry and immunofluorescence. Results Compared with the control group, the myelin sheath significantly reduced, and the expression levels of Olig2, GFAP and NG2 in CC were significantly higher in the demyelination group (P<0.01). The expression of NG2 in lateral septal nucleus (LSD) were higher than that in CC (P<0.05). The expression levels of Olig2 and GFAP in the remyelination group were significantly higher than that in the control group (P<0.05), but were significantly lower than that in the demyelination group (P<0.05). Conclusion Oligodendrocyte precursor cells cannot migrate to the site of injury in time. Sustained high level of astrocytes and reduction of Olig2 may be the influencing factors for myelin sheath repair disorder at the repair stage.

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