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慢性弓形虫感染小鼠小胶质细胞的活化与炎症因子的表达

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Microglial Activation and Inflammatory Cytokine Expression in the Brain of Chronic Toxoplasma gondii-infected Mice

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摘要 目的 探讨慢性弓形虫感染小鼠小胶质细胞的活化与炎症因子的表达。 方法 将30只ICR小鼠随机分成慢性弓形虫感染组和健康对照组，每组15只，感染组小鼠每只经口服感染弓形虫TgCtwh6株包囊30个，对照组喂饮等量生理盐水0.3 ml。感染后60 d，分离脑组织，应用免疫组化法分析小胶质细胞的数量和形态变化，逆转录PCR（RT-PCR）检测脑组织中白细胞介素?鄄1β（IL-1β）、IL-6和α肿瘤坏死因子（TNF-α）水平，免疫荧光标记和蛋白质印迹法（Western blotting）检测诱导型一氧化氮合酶（iNOS）的表达。 结果 免疫组化法检测结果显示，感染组小鼠的皮层区和海马区的离子钙接头分子（Iba-1）阳性细胞数（即小胶质细胞）（16.5±0.8和17.9±1.1）显著高于对照组（8.4±0.2和10.3±0.8）（P<0.05），且细胞呈明显活化状态，胞体增大，分支增多。RT-PCR结果显示，弓形虫感染组IL-1β、IL-6和TNF-α mRNA转录水平（0.862±0.169、0.407±0.158和0.305±0.073）均显著高于对照组（0.149±0.030、0.037±0.008和0.001±0.001）（P<0.05）。慢性弓形虫感染小鼠的脑组织中iNOS蛋白表达水平（0.252±0.164）显著高于对照组（0.043±0.004）（P<0.05）。免疫荧光标记结果显示，iNOS蛋白均由小胶质细胞表达。 结论 慢性弓形虫感染小鼠脑内小胶质细胞显著活化，IL-1β、IL-6和TNF-α等炎症因子和iNOS水平升高。

关键词： 刚地弓形虫 慢性感染 小胶质细胞 炎症因子

Abstract： Objective To investigate microglial activation and inflammatory cytokine expression in chronic Toxoplasma gondii infection. Methods Thirty mice were randomly divided into chronic T. gondii infection group and normal control group. Each mouse in infection group was infected orally with 30 cysts of the TgCtwh6 strain. Normal group received 0.3 ml normal saline. On the 60th day after infection， immunohistochemical staining was performed to assess the number of microglia and morphological change. The expression of inflammatory cytokines （IL-1β， IL-6， and TNF-α） was measured by RT-PCR. The expression of iNOS was determined by Western blotting and immunofluorescence. Results Immunohistochemistry analysis showed that the number of Iba-1 positive cells in the cortex and hippocampus of infection group （16.5±0.8 and 17.9±1.1） was higher than that of the control（8.4±0.2 and 10.3±0.8）（P<0.05）. Iba-1 positive cells （i.e. microglia） had larger cell bodies and ramified morphology. RT-PCR result indicated that mRNA level of IL-1β， IL-6， and TNF-α in infection group （0.862±0.169， 0.407±0.158， and 0.305±0.073） was significantly higher than that of the control （0.149±0.030， 0.037±0.008， and 0.001±0.001）（P<0.05）. The iNOS protein expression in infection group （0.252±0.164） was higher than that of the control（0.0433±0.004）（P<0.05）. Immunofluorescence demonstrated that iNOS protein released by activated microglia. Conclusion Chronic T. gondii infection caused micro-glial activation， which up-regulate the level of IL-1β， IL-6， TNF-α， and iNOS.

Keywords： Toxoplasma gondii Chronic infection Microglia Inflammatory cytokines

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