论著

细胞信号转导抑制剂介导Th1/Th2免疫偏移对血吸虫虫卵肉芽肿 的影响

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目的 观察酪氨酸蛋白激酶(TPK)、蛋白激酶C(PKC)和磷酯酰肌醇-3-激酶(P13-K)特异性抑制剂(分 别为tyrphostin-25、D-sphingosine和wortmannin)对日本血吸虫感染小鼠虫卵肉芽肿病变的影响。 并探讨其作用机制。 方法 于小鼠感染日本血吸虫后第35天起,经尾静脉分别注射3种信号转导分子抑制 剂,连续5 d。在小鼠感染后6和8wk,观察小鼠肝肉芽肿病变,并用ELISA夹心法和硝酸还原酶法分别测定 小鼠血清IFN-γ、IL-4和一氧化氮(NO)水平。结果 感染小鼠应用TPK和PKC抑制剂后均可显著抑制肝肉 芽肿病变,PKC抑制剂可使肉芽肿减少率达56.2%~63.4%(P<0.01)。PKC抑制剂主要抑制Th2细胞因 子IL-4的表达,其抑制率为34.1%和65.6%(P<0.01),而对NO水平的检测结果进一步证明了PKC抑制剂 对IL-4表达的抑制作用。 结论 在日本血吸虫感染早期应用PKC抑制剂干预T淋巴细胞信号转导可显著抑 制小鼠肝肉芽肿病变,其机制可能是由于抑制了Th2优势应答并介导Th2向Th1免疫反应偏移。

关键词 日本血吸虫 虫卵肉芽肿 细胞信号转导 Th1/Th2细胞因子 分类号

Effect of Inhibitors of Cell Signal Transduction on Egg Granuloma Formation in Mice Infected with Schistosoma japonicum

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Objective To observe the effect of signaling inhibitors of tyrosine-protein kinase (TPK), protein kinase C (PKC) and phosphatidylinositol-3-kinase (PI3-K) (tyrphostin-25, Dsphingosine and wortmannin, respectively) on the egg granuloma formation of Schistosama japonicum, and probe the mechanism of the effect. Methods Three signaling inhibitors were injected by tail vein of mice from the thirty-fifth day after infection for five successive days. The liver egg granuloma measurement was performed by histological examination and the kits of ELISA and NO assay were used for the quantitative determination of IFN-y, IL-4 and NO respectively in murine serum at 6 and 8 weeks after infection. Results The egg granuloma formation of liver tissue was significantly reduced by the specific inhibitors of TPK and PKC in vivo. The ratio of egg granuloma inhibition was up to 56.2% - 63.4% by the effects of PKC inhibitor Dsphingosine. The PKC inhibitor mainly inhibited the expression of IL-4 and the detection of NO level further demonstrated the inhibition. Conclusion The egg granuloma formation could be significantly inhibited by PKC inhibitor in the early stage of Schistosama japonicum infection in mice. These findings suggest that PKC inhibitor might inhibit the Th2 bias and mediate a deviation from Th2 to Th1. Key words Schistosoma japonicum egg granuloma singnal transduction Th1/Th2

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