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银杏酮酯对衰老大鼠海马炎症相关 细胞因子的调节作用

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中文摘要：目的：研究银杏酮酯(*Ginkgo biloba* extract 50,GBE50)对衰老模型大鼠海马促炎症细胞因子白介素-1*β*(interleukin-1 beta,IL-1*β*)、肿瘤坏死因子-*α*(tumor necrosis factor-alpha,TNF-*α*)和抗炎症细胞因子白介素-4(interleukin-4,IL-4)、白介素-10(interleukin-10,IL-10)的调节作用,探索GBE50对衰老动物中枢神经系统的保护机制。 方法：SD大鼠随机分为4组:正常组、模型组、GBE50组和EGB761组,采用*D*-半乳糖100 mg·kg^{-1}每日腹腔注射建立衰老大鼠模型,持续42 d,GBE50组和EGB761组分别在造模第21天开始给予60 mg·kg^{-1}剂量灌胃,共21 d。采用实时荧光定量PCR检测海马细胞因子IL-1*β* mRNA和TNF-*α* mRNA表达,免疫组织化学法检测海马细胞因子IL-1*β*和TNF-*α*蛋白表达,酶联免疫吸附试验测定海马细胞因子IL-4和IL-10蛋白含量。 结果：*D*-半乳糖可造成衰老模型大鼠海马促炎症细胞因子和抗炎症细胞因子的失调,GBE50和EGB761降低了海马IL-1*β* mRNA表达(*P*<0.05),减少了TNF-*α*和IL-1*β*的蛋白表达水平(*P*<0.01),上调了IL-10蛋白含量(*P*<0.01,*P*<0.05)。 结论：GBE50保护中枢神经系统的作用机制可能与GBE50改善中枢神经系统炎症有关。

中文关键词：衰老 银杏酮酯 促炎症细胞因子 抗炎症细胞因子

Regulating effect of *Ginkgo biloba* extract 50 on hippocampal inflammation-related cytokines in senile rats

Abstract:Objective: To investigate the regulating effect of *Ginkgo biloba* extract 50 (GBE50) on pre-inflammatory factors interleukin-1 beta (IL-1*β*), tumor necrosis factor-alpha (TNF-*α*) and anti-inflammatory factors interleukin-4 (IL-4), interleukin-10(IL-10) of hippocampus in senile rats, in order to explore the protective mechanism of GBE50 on central nervous system of senile animals. **Method:** SD rats were randomly divided into four groups: the normal group, the model group, the GBE50 group and the EGB761 group. Rats were intraperitoneally injected with 100 mg·kg^{-1} *D*-galactose every day for 42 days to establish the senile rat model. At the 21st day, the GBE50 group and the EGB761 group were orally administered with 60 mg·kg^{-1} for 21 days. IL-1*β* mRNA and TNF-*α* mRNA expressions were detected by real-time fluorescence quantitative PCR assay, IL-1*β* and TNF-*α* protein expressions were detected by immunohistochemistry, IL-4 and IL-10 protein contents were detected by ELISA. **Result:** *D*-galactose caused imbalance between pre-inflammatory factors and anti-inflammatory factors of hippocampus in senile rats, GBE50 and EGB761 reduced IL-1*β* mRNA expression (*P*<0.05) and TNF-*α* and IL-1*β* protein level (*P*<0.01) and up-regulated IL-10 protein content (*P*<0.01, *P*<0.05). **Conclusion:** The mechanism of GBE50 in protecting central nervous system is probably related to its effect in mitigating inflammatory of central nervous system.

keywords: senile GBE50 pre-inflammatory factor anti-inflammatory factor

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