

论著

扩展功能

本文信息

► [Supporting info](#)

► [PDF\(947KB\)](#)

► [\[HTML全文\]\(0KB\)](#)

► [参考文献](#)

服务与反馈

► [把本文推荐给朋友](#)

► [加入我的书架](#)

► [加入引用管理器](#)

► [复制索引](#)

► [Email Alert](#)

► [文章反馈](#)

► [浏览反馈信息](#)

相关信息

► [本刊中包含“活性部位,当归芍药散”的相关文章](#)

► 本文作者相关文章

· [胡增峣](#)

· [杨胜](#)

· [周文霞](#)

当归芍药散活性部位JD-3O对淀粉样 β 蛋白片段25-35抑制大鼠海马脑片CA1区长时程增强的改善作用

胡增峣, 杨胜, 周文霞*, 张永祥, 尚玮玮, 乔善义

(军事医学科学院毒物药物研究所, 北京 100850)

收稿日期 2007-1-11 修回日期 网络版发布日期 2007-7-10 接受日期 2007-3-29

摘要 目的 研究当归芍药散改善学习记忆能力的物质基础和作用机理。方法 细胞外微电极记录技术, 记录大鼠海马脑片CA1区群峰电位(PS)幅值和高频刺激诱发LTP后PS的增幅。结果 当归芍药散活性部位JD-3O(25, 50和100 mg·L⁻¹)的人工脑脊液灌流大鼠海马脑片, 其CA1区的PS幅值无明显变化。用相同浓度的JD-3O孵育海马脑片90 min以上并持续灌流, 其CA1区高频刺激后的PS增幅与空白对照组相比无明显差异。而用A β ₂₅₋₃₅ 200 nmol·L⁻¹处理的海马脑片CA1区高频刺激后的PS增幅受到明显抑制; 若同时给予A β ₂₅₋₃₅和上述浓度的JD-3O处理海马脑片, CA1区高频刺激后的PS增幅较A β ₂₅₋₃₅组升高, 其中JD-3O 100 mg·L⁻¹组的PS增幅达到正常对照组水平。提示JD-3O对正常海马脑片CA1区的基础突触传递和LTP没有影响, 但可改善A β ₂₅₋₃₅所抑制的LTP。结论 JD-3O可改善神经突触可塑性, 抗A β 对LTP的抑制作用可能是其益智机制之一。

关键词 活性部位, 当归芍药散, 淀粉样 β 蛋白, 海马, 长时程增强, 微电极

分类号 [R282.5, R971](#)

JD-30, an active constituent extracted from Danggui Shaoyao San, ameliorates amyloid beta-protein fragment 25-35 induced inhibition of long-term potentiation of CA1 area in rat hippocampal slices

HU Zeng-Yao, YANG Sheng, ZHOU Wen-Xia*, ZHANG Yong-Xiang, SHANG Wei-Wei, QIAO Shan-Yi

(Institute of Pharmacology and Toxicology, Academy of Military Medical Sciences, Beijing 100850, China)

Abstract

AIM To study the material base and mechanism of cognition enhancing effect of Danggui Shaoyao San (DSS, also named Toki-shakuyaku-san in Japanese). **METHODS** By using extracellular microelectrode recording technique, population spike (PS) and long-term potentiation(LTP) of CA1 area in hippocampal slices were induced and recorded.

RESULTS Perfused with JD-30(25, 50 and 100 mg·L⁻¹), an active constituent extracted from DSS, had no effect on PS amplitude of CA1 area in hippocampal slices. Incubated and perfused with the same concentrations of JD-30 had no effect on PS amplification of CA1 area after high frequency stimulation. Treatment with A β ₂₅₋₃₅ 200 nmol·L⁻¹ significant depressed PS amplification of CA1 area, and the inhibition by A β ₂₅₋₃₅ was ameliorated by JD-30. Especially, JD-30 100 mg·L⁻¹ retrieved PS amplification to the normal group level. **CONCLUSION** JD-30 may improve synaptic plasticity, and it is possibly one of the mechanisms for its cognition enhancement action to ameliorate the inhibition of A β on LTP.

Key words [active fraction](#), [Danggui Shaoyao San](#), [amyloid beta-protein](#), [hippocampus](#), [long-term potentiation](#), [microelectrodes](#)

DOI: