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黄芪甲苷对氧化低密度脂蛋白诱导内皮祖细胞炎症损伤的保护作用

Protective Effects of Astragaloside on Function of EPCs Damaged by ox-LDL

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中文关键词： 黄芪甲苷 内皮祖细胞 氧化低密度脂蛋白 炎症

英文关键词:astragaloside ndothelial progenitor cells(EPCs) oxidative low density lipoprotein(ox-LDL) inflammatory

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中文摘要:

目的 观察黄芪甲苷对氧化低密度脂蛋白(oxidative low density lipoprotein，ox-LDL)介导的内皮祖细胞(endothelial progenitor cells，EPCs)炎症损伤的保护作用并探讨其可能机制。方法 密度梯度离心法获取外周血单个核细胞，贴壁法培养EPCs。培养7 d后，收集贴壁细胞并随机分为对照组、ox-LDL组(100 μg·mL^{-1})及黄芪干预组(ox-LDL 100 μg·mL^{-1}加黄芪甲苷，浓度分为2，10和50 μg·mL^{-1})，干预24 h后分别采用Matrigel体外成血管试验、Transwell小室法、黏附能力测定实验及细胞计数试剂盒(Cell Counting Kit-8，CCK-8)观察ox-LDL对EPCs成血管能力、迁移能力、黏附能力及增殖能力的影响，并取各组细胞培养上清液行白细胞介素-6(IL-6)和肿瘤坏死因子α(TNF-α)含量检测。结果 ox-LDL损伤后，外周血EPCs的成血管能力、迁移能力、黏附能力及增殖能力显著受损，伴随细胞上清液IL-6及TNF-α水平显著升高；黄芪甲苷干预24 h后，显著改善了EPCs的成血管、迁移、黏附及增殖能力，且黄芪甲苷各组IL-6及TNF-α水平显著降低。结论 黄芪甲苷对ox-LDL损伤后EPCs的细胞生物学功能有显著保护作用，其机制可能与抗炎症损伤有关。

英文摘要:

OBJECTIVE To observe the protective effects of astragaloside on inflammatory injury induced by oxidative low density lipoprotein (ox-LDL) in endothelial progenitor cells(EPCs), and find the potential mechanisms. METHODS Total mononuclear cells(MNCs) were isolated from peripheral blood of healthy young human volunteers by ficoll density gradient centrifugation, and plated on fibronectin-coated culture dishes. After incubation for 7 days, attached cells will be collected and randomized into five groups: control group, ox-LDL-intervented group, and three astragaloside-intervented groups which were respectively added with different concentrations of astragaloside (2, 10 and 50 μg·mL^{-1}) and 100 μg·mL^{-1} ox-LDL. After intervention for 24 hours, the capacities for EPCs vasculogenesis, migration, adherence, as well as proliferation separatively were evaluated and the levels of IL-6 and TNF-α in the culture supernate of the five groups were measured. RESULTS Compared with the control group, the capacities for EPCs vasculogenesis, migration, adherence, as well as proliferation were impaired and the levels of IL-6 and TNF-α were obviously elevated in the ox-LDL-intervented group ($P<0.01$). In contrast, these capacities as well as IL-6 and TNF-α levels were improved in astragaloside-intervented groups. CONCLUSION Astragaloside can protect the EPCs capacities of vasculogenesis, migration, adherence, and proliferation which would be injured by ox-LDL. The potential mechanism might be related to its anti-inflammatory features.