基于MAPK/STAT3通路探讨人参皂苷F1对变应性鼻炎的改善作用及机制Δ

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摘　要　目的　基于丝裂原激活的蛋白激酶(MAPK)/信号转导及转录活化因子3(STAT3)通路探讨人参皂苷F1(GF1)对变应性鼻炎(AR)大鼠的改善作用及潜在机制。**方法**　将雄性SD大鼠分为对照组(15只)和造模组(55只),其中造模组大鼠采用卵白蛋白基础致敏+局部激发的方式构建AR大鼠模型。将造模成功的大鼠随机分为AR组、低剂量GF1组[2.5 mg/(kg·d)]、高剂量GF1组[5 mg/(kg·d)]、氯雷他定组[阳性对照,1.5 mg/(kg·d)]、高剂量GF1+MAPK激活剂组[5 mg/(kg·d)GF1+0.2 nmol C16-PAF],每组10只;另从对照组中随机选取大鼠10只,作为空白对照组。各组大鼠经鼻腔滴注或(和)灌胃相应药液或生理盐水,每天1次,连续28 d。末次给药后,观察大鼠AR症状并进行鼻炎症状评分,观察其鼻黏膜组织病理改变,检测其鼻腔灌洗液中炎症细胞数量,血清中炎症因子[白细胞介素4(IL-4)、IL-17、γ干扰素(IFN-γ)]和免疫球蛋白E(IgE)水平,鼻腔黏膜组织中杯状细胞密度、肥大细胞数量以及MAPK/STAT3通路相关蛋白的表达情况。**结果**　与空白对照组比较,AR组大鼠鼻黏膜组织上皮层病理损伤明显,可见间质水肿、炎症细胞浸润等病理改变;其鼻炎症状评分,巨噬细胞、淋巴细胞、嗜酸性粒细胞、中性粒细胞、肥大细胞数量和杯状细胞密度,IL-4、IL-17、IgE水平,以及p38 MAPK、STAT3蛋白的磷酸化水平均显著升高或增加,IFN-γ水平显著降低($P<0.05$)。与AR组比较,各药物组大鼠鼻黏膜组织上皮层病理损伤均有所恢复,各定量指标均显著改善,且高剂量GF1组、氯雷他定组的改善均较低剂量GF1组显著($P<0.05$);C16-PAF可显著逆转GF1对各定量指标的改善作用($P<0.05$)。**结论**　GF1可改善AR大鼠的鼻炎症状,减轻鼻黏膜组织炎症反应;上述作用可能与其抑制MAPK/STAT3通路激活有关。

关键词　人参皂苷F1;变应性鼻炎;炎症反应;MAPK/STAT3通路

Improvement effect and mechanism of ginsenoside F1 on allergic rhinitis rats based on the MAPK/STAT3 pathway

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ABSTRACT　OBJECTIVE To explore the improvement effect and potential mechanism of ginsenoside F1 (GF1) on allergic rhinitis (AR) rats based on the mitogen-activated protein kinase (MAPK)/signal transducer and activator of transcription 3 (STAT3) pathway. **METHODS** Male SD rats were divided into a control group ($n=15$) and a modeling group ($n=55$). The AR rat model was established by basic sensitization with ovalbumin followed by local challenge. The successfully modeled rats were randomly assigned to the AR group, low-dose GF1 group [2.5 mg/(kg·d)], high-dose GF1 group [5 mg/(kg·d)], loratadine group (positive control, 1.5 mg/(kg·d)], and high-dose GF1+MAPK activator group [5 mg/(kg·d) GF1+0.2 nmol C16-PAF], with 10 rats in each group. Another 10 rats from the control group were randomly selected as the blank control group. Rats in each group received corresponding drug solutions or normal saline via intranasal instillation and/or oral gavage once daily for 28 consecutive days. After the final administration, AR symptoms were observed and rhinitis symptom scores were assessed. Histopathological changes of the nasal mucosa were examined. The number of inflammatory cells in nasal lavage fluid, serum levels of inflammatory cytokines [interleukin-4 (IL-4), IL-17, interferon-γ (IFN-γ)] and immunoglobulin E (IgE), as well as the density of goblet cells, the number of mast cells, and the expression of MAPK/STAT3 pathway-related proteins in nasal mucosal tissues were measured. **RESULTS** Compared with the blank control group, the AR group exhibited marked pathological damage to the nasal mucosal epithelium, characterized by interstitial edema and inflammatory cell infiltration. The rhinitis symptom score, the number of macrophages, lymphocytes, eosinophils, neutrophils and mast cells, the density of goblet cells, the levels of IL-4, IL-17 and IgE, as well as the protein phosphorylation levels of p38 MAPK and STAT3 were significantly increased or elevated, while the level of IFN-γ was significantly decreased in the AR group ($P<0.05$). Compared with the AR group, all administration groups showed recovery of nasal mucosal epithelial pathological damage and significant improvements in all quantitative indicators, with the high-dose GF1 group and loratadine group

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