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Inhibitory effects of salidroside on inflammatory responses in rat alveolar macrophages

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The genus Rhodiola is an important herb used for thousands of years in Traditional Chinese Medicine for treating various kinds of diseases, including inflammation-related diseases such as pneumonia, chronic bronchitis, asthma, and rheumatoid arthritis. Salidroside (p-hydroxyphenethyl-beta-d-glucoside, C14H20O7, molecular weight 300.30), which is one of the most potent ingredients of the genus Rhodiola, has been reported to have a broad spectrum of pharmacological properties. Recent study indicates an anti-inflammatory role for salidroside in lipopolysaccharide (LPS)-induced acute lung injury (ALI), but the mechanisms of anti-inflammatory action are not clear. In the present study, anti-inflammatory activity of salidroside was analyzed in vitro using LPS-induced inflammatory reactions in NR8383 cells (a rat alveolar macrophage cell line) and primary rat alveolar macrophages. Since tumor necrosis factor-α (TNF-α), interleukin (IL)-6, nitric oxide (NO), and prostaglandin E2(PGE2) play key roles in inflammation, concentration of those inflammatory mediators in the culture supernatant were measured by the Griess reaction for NO and Enzyme linked immunosorbent assay (ELISA) for other inflammatory mediators. QRT-PCR was used to measure the mRNA expression of TNF-α, IL-6,inducible NO synthase (iNOS), and cyclooxygenase-2(COX-2), and the protein levels of iNOS, COX-2, p65, p-p65, IκB-α, p-IκB-α, JNK, p-JNK, ERK, p-ERK, P38, P-P38 were analyzed by western blot. Electrophoretic mobility shift assays (EMSAs) and reporter gene assays were used to evaluate DNA binding and transcriptional activities of NF-κB. Chromatin immunoprecipitation (ChIP) analysis was used to detect the binding of p65 to the iNOS promoter. NF-κB nuclear translocation was analyzed by western blot and immunofluorescence. Salidroside significantly inhibited LPS-stimulated production of NO and PGE2 in NR8383 cells by suppressing the expression of iNOS and COX-2. Salidroside also suppressed TNF- and IL-6 in LPS-stimulated NR8383 cells at the mRNA level, and then reduced the release of TNF-α and IL-6 induced by LPS. Salidroside inhibited NF-κB transcriptional activity by blocking the formation of NF-κB–DNA complexes, leading to down-regulation of the NF-κB target genes in LPS stimulated NR8383 cells, and also inhibited LPS-induced nuclear translocation of NF-κB. Salidroside inhibited the LPS-induced activation of IκB and MAPKs. Similar findings were observed with primary macrophages derived from rat lung. In conclusion, our results suggest that salidroside can block LPS stimulated inflammatory responses in rat alveolar macrophages, and may be used as a potential anti-inflammatory agent for the prevention and treatment of inflammatory diseases.

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