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• 英文摘要	Dysfunction of the endothelium contributes to pathological conditions of the arterial wall, such as atherosclerosis [1]. Clinical evidences indicate that the expression of cytokines in human aortic intima during atherogenesis and elevated systemic parameters in patients with coronary atherosclerosis [2-4]. Therefore, proper control of cytokines release might be beneficial on limitation of atherogenic process. Chronic inflammation is well known to be involved in vascular injuries such as atherosclerosis in which interferon-gamma may be involved. Expression of Interferon-gamma (IFN-gamma) in atherosclerotic lesions from both clinical samples as well as in preclinical rodent atherosclerosis models has been demonstrated [5-7]. In addition, IFN-gamma aggravates atherosclerosis in apoE-deficient mice [8]. These results confirm that IFNgamma is a major cytokine that contributes to atherogenesis. Effective blockage of IFN-gamma-induced inflammatory responses may provide a provital role in anti-athersclerosis. Salvia miltiorrhiza has been used in traditional Chinese medicine for thousands of years and is now widely used for treatment of cardiovascular diseases. Salvianolic acid B (Sal B), an active component of Salvia miltiorrhiza, was reported to exert anti-inflammatory and anti-oxidative properties. Early studies showed Sal B significantly inhibited vascular cell adhesion molecule-1 (VCAM-1) and intercellular cell adhesion molecule-1(ICAM-1) in TNF-alpha-treated human aortic endothelial cells [9]. TNF-alpha-increased endothelial permeability, which might implicate in inflammation process, was attenuated by pretreatment with Sal B [10]. Salvianolic acids protected endothelial cells against damage induced by cholestane-3beta-5alpha-6beta-triol [11]. These results elicit Sal B has anti-inflammatory properties and may explain their		

anti-atherosclerotic effects. However, the detailed signaling pathways and downstream effects on regulation of IFN-gamma-related responses in ECs that contribute to Sal B-induced atheroprotection remain to be defined.Salvianolic acid B (Sal B) is used reported to be the active component from traditional Chinese medicine, Salvia miltiorrhiza, that exert anti-inflammatory and anti-oxidative properties. Sal B possesses anti-inflammatory properties that are believed to contribute to its therapeutic effects. In this study, we determined that Sal B inhibits the IFN-gamma-induced JAK-STAT1 signaling pathway that results in suppression of downstream inflammation-associated genes. Several lines of evidence support this notion. First, Sal B inhibited the IFN-gamma-induced responses by suppressing Jak2 and Stat1 phosphorylation. Second, it suppressed the IFN-gamma-induced IP-10 promoter activity. Third, Sal B inhibited the IFN-gamma-induced IP-10 protein release. Fourth, IFN-gamma-induced CXC chemokines gene expressions including IP-10, Mig, and I-TAC, ICAM-1, were suppressed by Sal B pretreatment. Fifth, Sal B treatment alone induced cytokine inhibition regulator proteins expression including PIAS-1 and SOCS-1. These results provide direct evidence that Sal B exerts atheroprotective effects on ECs by suppressing IFN-gamma-induced JAK-STAT1 activation through PIAS-1 and SOCS-1.