For Research Use Only Astragaloside



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Catalog Number: CM12393

产品信息

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CAS号:

17429-69-5

分子式: C₂₈H₃₂O₁₇

MMP|ERK|JNK

主要通路: MAPK信号通路|蛋白酶体

分子量: 640.54

DMSO:390 mg/mL, Sonification is recommended.



体外活性

Astragaloside IV (10, 20, 40 ng/mL) inhibits NSCLC cell growth, whereas low concentrations of astragaloside IV (1, 2.5, 5 ng/mL) has no obvious cytotoxicity on cell viability. Moreover, combined treatment with astragaloside IV significantly increases chemosensitivity to cisplatin in NSCLC cells. On the molecular level, astragaloside IV co-treatment significantly inhibits the mRNA and protein levels of B7-H3 in the presence of cisplatin. Astragaloside IV inhibits the viability and invasive potential of MDA-MB-251 breast cancer cells, suppresses the activation of the mitogen activated protein kinase (MAPK) family members ERK1/2 and JNK, and downregulates matrix metalloproteases (MMP)-2 and -9.

Astragaloside IV (10, 20 mg/kg, p.o.) exhibits a potent ability to prevent cognitive deficits induced by transient cerebral ischemia and reperfusion. Astragaloside IV (10 mg/kg) and Astragaloside IV (20 mg/kg) can significantly decrease the levels of these cytokines compared to the Model group. Astragaloside IV significantly inhibits the level of TLR4 and its downstream proteins, suggesting that both MyD88-dependent and -independent pathways play important roles in the anti-inflammatory effects of Astragaloside IV. Astragaloside IV attenuates NLRP3 and cleaved-caspase-1 expression, and reduces Iba1 protein expression[1]. In the mice model, the high-dose astragaloside IV group has a significant increase in the 48-hour survival rate [60% (9/15) vs 13.3% (2/15), P < 0.05], significant reductions in the serum ALT and AST levels (P < 0.01), and significant reductions in liver histopathological indices and the degree of apoptosis of hepatocytes (P < 0.01), as well as a significant reduction in the content of MDA in liver homogenate (P < 0.01) and a significant increase in the activity of SOD.

动物实验

Transient cerebral ischemia and reperfusion is prepared by BCCAO, as BCCAO is considered an ideal model to study transient cerebral ischemia and reperfusion injury-mediated inflammatory response. Mice are randomLy divided into the Sham, Model, Astragaloside IV (10 mg/kg) and Astragaloside IV (20 mg/kg) treatment groups. The Astragaloside IV treatment groups are intragastrically administered 7 days before the surgery and terminated on the day of sacrifice. On the day of the surgery, Astragaloside IV is administrated 2 h prior to ischemia. The Sham-operated and Model groups are treated with distilled water. After the mice are anesthetized with an intraperitoneal injection of chloral hydrate (350 mg/kg), the bilateral common carotid arteries are exposed and carefully separated with a small ventral neck incision and occluded twice (20 min each) with ligated surgical silk as described previously with minor modifications. There is a 10 min reperfusion period between the two occlusion periods (ischemia 20 min? reperfusion 10 min? ischemia 20 min). Sham-operated mice are subjected to the same surgical operation without the surgical silk ligation. Mouse body temperature is maintained at 37±0.5°C during the surgery with heating equipment until recovery from the anesthesia. recovery from the anesthesia.

细胞实验

Cell viability is determined by CCK-8 assay. To be brief, cultured NSCLC cells are seeded into 96-well plates at the density of 4×104 (cells/well). Then 10 $\,\mu$ L?well CCK8 solution is added and incubated in dark at 37°C for another 2 h. The absorbance is determined with the wavelength of 490 nm.

Astragaloside, one of the main active ingredients in Astragalus membranaceus.

Powder: -20°C for 3 years | In solvent: -80°C for 2 years