For Research Use Only Forskolin



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Catalog Number: CM00273

产品信息

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CAS号: 66575-29-9

分子式: $C_{22}H_{34}O_{7}$

主要靶点:

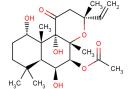
Autophagy|Adenylyl cyclase|AChR|FXR

主要通路: 神经科学|神经科学|代谢|自噬

分子量: 410.5

溶解度:

10% DMSO+40% PEG300+5% 10% DM30+40% PEQ300+39 Tween 80+45% Saline:3 mg/mL (7.31 mM);Ethanol:15 mg/mL (36.5 mM);DMSO:55 mg/mL (133.98 mM);H2O:Insoluble



靶点活性

体外活性

体内活性

动物实验

细胞实验

储存

Adenylyl cyclase:0.5 µ M (cell free)

方法: 大鼠肾上腺髓质嗜铬瘤细胞 PC12 用 Forskolin (0.01-10 μ M) 处理 3-48 h, 使用 MTT 方法检测细胞生长抑制情况。结果:用 10 μ M Forskolin 处理后,细胞活力迅速下降,处理 6 h 后细胞活力下降为 88.4%,处理 48 h 后细胞活力下降为 60.5%。[1] 方法: 人骨髓瘤细胞 U266、H929、INA-6、RPMI 8226 和 OPM-2 用 Forskolin (1-100 μ M) 处理 72 h, 使用 Flow Cytometry 方法检测细胞死亡情况。结果:Forskolin 剂量依赖性诱导人骨髓瘤细胞死亡,其中 U266、OPM-2 和 INA-6 比 H929 和 RPMI 8226 细胞更敏感。[2] 方法: 人 IL-2 依赖性白血病细胞 Kit 225 和人白血病细胞 MT-2 用 Forskolin (1-100 μ M) 处理 20 min,使用 EUSA 方法测定 cAMP 浓度。结果:Forskolin 诱导 cAMP 水平上调,在 50-100 μ m 之间达到最大水平。[3]

方法:为检测体内抗肿瘤活性,将 Forskolin (4-5 mg/kg in PBS/DMSO solution (15:1)) 腹腔注射给携带鼠多发性骨髓瘤肿瘤 MOPC315 的 BALB/c nude 小鼠,在肿瘤细胞注射后的第 2/4/6 天给药。结果:所有小鼠最终都发生了肿瘤,但 Forskolin 显著延缓了体内肿瘤的生长。提高 cAMP 的化合物可能在治疗多发性骨髓瘤方面具有治疗潜力。[4]方法:为研究 Forskolin 对糖尿病条件下视网膜炎症的影响,将 Forskolin (50 mg/kg) 灌胃给药给 STZ 诱导糖尿病模型的 C57BL/6 小鼠,每周一次,持续十二周。结果:与正常对照组相比,糖尿病对照组和 Forskolin 治疗组的视网膜葡萄糖浓度均增加,但由于葡萄糖转运蛋白 1表达下调,Forskolin 处理组仅为糖尿病对照组的约 68.06%。与正常对照组相比,Forskolin 治疗组和糖尿病对照组的 ICAM?1 和 TNF-a 表达上调,但 Forskolin 处理组的这两种炎症因子表达水平分别为糖尿病对照的 68.75% 和 75.37%。[5]

Forskolin was dissolved in dimethyl sulfoxide (DMSO) and injected intraperitoneally into neonatal mice at postnatal days 4 (P4) and 5 (P5). Mice injected with DMSO served as the controls. The treated mice were euthanized at P6, and their retinas were isolated for whole-mount immunohistochemistry (IHC). We first tested the effect of different concentrations of forskolin on the survival rate and retinal vasculature and determined the optimal concentration, 1.0 µ g/50 µ L (0.3 mg/kg) at P4 and 1.5 µ g/50 µ L (0.5 mg/kg) at P5, used to compare the retinal vascular phenotypes between WT mice and Mrp4-deficient mice [4]. After acclimatization for 2 weeks, animals were randomly divided into four groups of eight rats each and treated for six consecutive weeks as follows: The first group was treated with CCl4 (50% CCl4/corn oil; 0.5 mL·kg?1, i.p.) twice a week to induce liver fibrosis. The second group was given forskolin only at a dose of 10 mg·kg?1, i.p., dissolved in a DMSO/saline solution (1:49) five times a week. The third group was given both CCl4 and forskolin. The dose of forskolin used here was based on the results of our preliminary study. The fourth group served as the normal control, receiving vehicles only. At 24 h after the last injection, blood samples were collected from the retro - orbital plexus after light anesthesia with sodium pentobarbital (50 mg·kg?1, i.p.). Serum was separated by centrifugation at 3000× g for 10 min and was used for the assessment of liver functions. Rats were killed by cervical dislocation, and livers were removed and weighed. A portion of liver tissue was washed and homogenized to obtain a 20% (w·v?1) homogenate, which was used for assessment of oxidative stress, inflammatory and fibrogenic markers. Another portion was placed in formalin for immunohistochemical and histopathological analyses. The remainder was stored at ?80°C, together with the 20% homogenate, until needed [5]. euthanized at P6, and their retinas were isolated for whole-mount immunohistochemistry (IHC). We first

Kit 225 or MT-2 cells were treated with 1, 5, 10, 20, 50, or 100 $\,\mu$ M Forskolin for 20 min at 37 °C. Cells were lysed and clarified by centrifugation, and the concentration of cAMP was detected by direct cAMP ELISA. Optical density was measured at 405 nm, and the concentration of intracellular cAMP was calculated using a weighted four parameter logistic curve according to the manufactures instructions [2].

keep away from direct sunlight,keep away from moisture,store at low temperature | Powder: -20°C for 3 years | In solvent: -80°C for 1 year | Shipping with blue ice.