

Emodin (Emodol; Frangula Emodin)

Catalog No: tcsc1412



Available Sizes

Size: 50mg

Size: 100mg

Size: 200mg



Specifications

CAS No:

518-82-1

Formula:

$C_{15}H_{10}O_5$

Pathway:

Autophagy;Stem Cell/Wnt;Cell Cycle/DNA Damage

Target:

Autophagy;Casein Kinase;Casein Kinase

Purity / Grade:

>98%

Solubility:

DMSO : 9.4 mg/mL (34.78 mM; Need ultrasonic and warming)

Alternative Names:

Frangula emodin

Observed Molecular Weight:

270.24

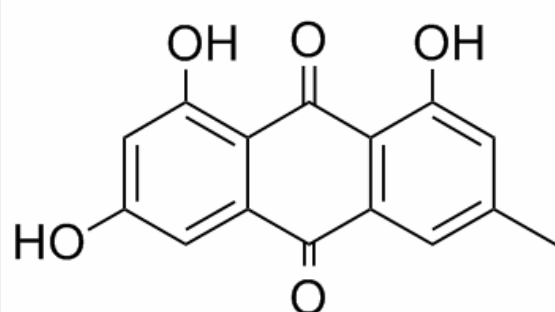
Product Description

Emodin is a broad-spectrum anticancer agent. Emodin inhibits **casein kinase II** (CKII) activity with **IC₅₀** of 2 μ M.

IC50 & Target: IC50: 2 μ M (CKII)^[1]

In Vitro: Emodin, an anthraquinone derivative, selectively inhibits casein kinase II(CKII), a Ser/Thr kinase, as a competitive inhibitor. Emodin inhibits CKII activity with IC₅₀ of 2 μ M, which is two to three orders of magnitude lower than those against the other kinases. Enzyme kinetic assays show that Emodin inhibits CKII activity as a competitive inhibitor against ATP with K_i of 7.2 μ M^[1]. Emodin is a broad-spectrum inhibitory agent of cancer cells, including leukemia, lung cancer, human tongue squamous cancer, colon cancer, gallbladder cancer, pancreatic cancer, breast cancer, human cervical cancer and hepatic carcinoma cells. Emodin inhibits A549, HepG2, OVCAR-3, HeLa and Madin-Darby Canine Kidney (MDCK) cells with IC₅₀ of 19.54, 12.79, 25.82, 12.14 and 5.81 μ g/mL. The anticancer mechanisms of Emodin are involved in many biological pathways, such as casein kinase II and ERK1/2^[2]. Emodin is applied as a Reactive oxygen species (ROS) generator in combination with cisplatin in T24 and J82 human bladder cancer cells. Emodin kills T24 and J82 cells in the dose-dependent and time-dependent manner, and it is less toxic to HCV-29 cells. The concentration of 20 and 15 μ M is selected as appropriate doses for investigating chemotherapeutic sensitivity of T24 and J82 cells at 24 h, respectively^[3].

In Vivo: Mice treated with Emodin (50 mg/kg) and Cisplatin (1 mg/kg) have significantly smaller tumors than those from the other groups. In addition, no notable differences on the body weight loss are observed among groups and no obvious necrosis and abnormality are observed in the sections of liver, kidney and heart. These results demonstrate that Emodin/cisplatin co-treatment can significantly suppress tumor growth in vivo with no distinct side effects. Consistent with in vitro experiment, TUNEL assay shows that Emodin/cisplatin combination significantly increased cell apoptosis in xenograft tumors. Emodin/Cisplatin co-treatment group also has lower MRP1 expression than the other groups^[3].



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