

Galangin Catalog No: tcsc5619

Available Sizes

Size: 10mg

Size: 25mg

Size: 50mg

Size: 100mg

Specifications

CAS No:

548-83-4

Formula:

 $C_{15}H_{10}O_{5}$

Pathway: Autophagy;Metabolic Enzyme/Protease

Target:

Autophagy;Cytochrome P450

Purity / Grade:

>98%

Solubility:

DMSO : ≥ 36 mg/mL (133.21 mM)

Alternative Names:

Norizalpinin;3,5,7-Trihydroxyflavone

Observed Molecular Weight: 270.24

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Product Description

Galangin is an agonist/antagonist of the arylhydrocarbon receptor, and also shows inhibition of **CYP1A1** activity.

In Vitro: Galangin inhibits the catabolic breakdown of DMBA, as measured by thin-layer chromatography, in a dose-dependent manner. Galangin also inhibits the formation of DMBA-DNA adducts, and prevents DMBA-induced inhibition of cell growth. Galangin causes a potent, dose-dependent inhibition of CYP1A1 activity, as measured by ethoxyresorufin-O-deethylase activity, in intact cells and in microsomes isolated from DMBA-treated cells. Analysis of the inhibition kinetics by double-reciprocal plot demonstrates that galangin inhibits CYP1A1 activity in a noncompetitive manner. Galangin causes an increase in the level of CYP1A1 mRNA, indicating that it may be an agonist of the aryl hydrocarbon receptor, but it inhibits the induction of CYP1A1 mRNA by DMBA or by 2,3,5,7-tetrachlorodibenzo-p-dioxin (TCDD). Galangin also inhibits the DMBA- or TCDD-induced transcription of a reporter vector containing the CYP1A1 promoter^[1]. Galangin treatment inhibits cell proliferation and induced autophagy (130 μM) and apoptosis (370 μM). In particular, galangin treatment in HepG2 cells causes (1) an accumulation of autophagosomes, (2) elevated levels of microtubule-associated protein light chain 3, and (3) an increased percentage of cells with vacuoles. p53 expression is also increased. The galangin-induced autophagy is attenuated by the inhibition of p53 in HepG2 cells, and overexpression of p53 in Hep3B cells restored the galangin-induced higher percentage of cells with vacuoles to normal level^[2].



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