Product Data Sheet

Product Name: Vitexicarpin (Casticin)
Cat. No.: GN10282

Chemical Properties

Cas. No.	479-91-4		
Chemical Name	5-hydroxy-2-(3-hydroxy-4-methoxyphenyl)-3,6,7-trimethoxychromen-4-one		
SMILES	COC1=C(C=C(C=C1)C2=C(C(=O)C3=C(C(=C(C=C3O2)OC)OC)O)OC)O		
Formula	$C_{19}H_{18}O_8$	M.Wt	374.35
Solubility	≥ 37.4mg/mL in DMSO	Storage	4°C, protect from light
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Caution: Product has not been fully validated for medical applications. For research use only.
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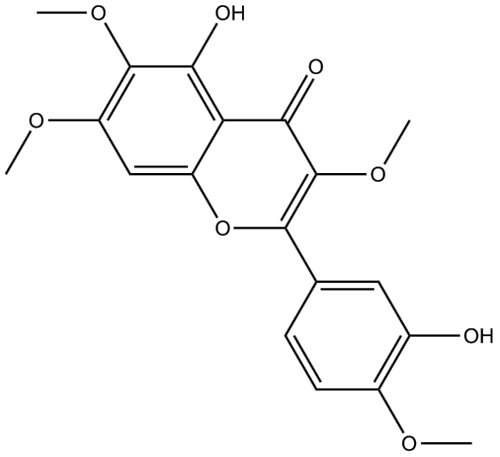
Structure



Protocol

Cell experiment [1]:

Cell lines	A549 cells
Preparation Method	A549 cells were cultured in RPMI-1640 medium supplemented with 10% fetal bovine serum (FCS), 100U/ml penicillin, and 100μg/ml streptomycin in a humidified incubator at 37°C and 5% CO_2. Cells were seeded in 96-well plates at a density of 5×10^3 cells/well at 100μl per well and treated with different concentrations of Vitexicarpin (0, 10, 20, 30, 40, and 50μM). After 24, 48, and 72 hours of incubation, 10μl of CCK-8 reagent was added to each well, and incubation was continued for 1 or 2 hours. Then, the absorbance value at 490nm was measured for each well.

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Reaction Conditions	0, 10, 20, 30, 40, and 50μM; 24, 48, and 72h
Applications	Vitexicarpin treatment inhibited the proliferation of A549 cells in a concentration- and time-dependent manner.

Animal experiment [2]:

Animal models	BALB/c nude mice
Preparation Method	Five-week-old BALB/c nude mice were maintained in a standard environment with ad libitum access to diet and water. 5×10^6 S18 cells were subcutaneously injected into the left side of the back of each mouse (3 mice per group). When the tumor volume reached approximately $100mm^3$, mice were randomly divided into control and vitexin treatment groups. Vitexicarpin was diluted in 10% DMSO in normal saline, and Vitexicarpin was injected intraperitoneally at a dose of 40mg/kg daily for 18 days; the control group was injected with normal saline only. Tumor size was measured and recorded every 3 days using a vernier caliper.
Dosage form	40mg/kg/day for 18 days; i.p.
Applications	Vitexicarpin treatment significantly reduced tumor growth in the S18 cell-xenograft mouse models.

References:

[1] Wei J, Lei G, Chen Q, et al. Casticin inhibits proliferation of non-small cell lung cancer cells through regulating reprogramming of glucose metabolism[J]. Phytomedicine, 2025, 136: 156278.
[2] Liu J, Yang J, Hou Y, et al. Casticin inhibits nasopharyngeal carcinoma growth by targeting phosphoinositide 3-kinase[J]. Cancer cell international, 2019, 19(1): 348.

Background

Product Data Sheet

Vitexicarpin (Casticin), a non-isotopic tetramethoxyflavone with three phenyl rings, is a phosphatidylinositol 3-kinase (PI3K) inhibitor with high cytotoxicity [1]. Vitexicarpin can down-regulate the mRNA and protein levels of DNA methyltransferase 1(DNMT1) and induce DNA damage by binding to topoisomerase IIα[2]. Vitexicarpin has been widely used as an anticancer agent to inhibit the malignant progression of cancer cells in diverse tumor cell models[3].

In vitro, Vitexicarpin significantly inhibited the proliferation of K562, HL-60, and Kasumi-1 cells with IC_{50} values of 5.95μM, 4.82μM, and 15.56μM, respectively [4]. Treatment with Vitexicarpin at 8μM for 24h significantly induced apoptosis and G2/M phase arrest in S18 cells[5]. Treatment of MGC803 cells with 30μM Vitexicarpin for 72h significantly reduced cell viability, increased protein expression and mRNA levels of RECK, and decreased global DNA methylation levels[6].

In vivo, Vitexicarpin treatment (20mg/kg/day) via intragastric administration for 2 weeks significantly reduced liver injury and liver fibrosis induced by bile duct ligation in mice[7]. Vitexicarpin(0.4mg/kg) was injected intraperitoneally once every two days for 18 days, which significantly reduced the tumor volume and weight of SCC-4 cell xenograft mice without affecting the body weight of the mice[8].

References:
[1] Ramchandani S, Naz I, Lee J H, et al. An overview of the potential antineoplastic effects of casticin[J]. Molecules, 2020, 25(6): 1287.
[2] Carbone K, Gervasi F, Kozhamzharova L, et al. Casticin as potential anticancer agent: recent advancements in multi-mechanistic approaches[J]. Frontiers in Molecular Biosciences, 2023, 10: 1157558.
[3] Ding X J, Cai X M, Wang Q Q, et al. Vitexicarpin suppresses malignant progression of colorectal cancer through affecting c-Myc ubiquitination by targeting IMPDH2[J]. Phytomedicine, 2024, 132: 155833.
[4] Shen J K, Du H, Yang M, et al. Casticin induces leukemic cell death through apoptosis and mitotic catastrophe[J]. Annals of hematology, 2009, 88(8): 743-752.
[5] Liu J, Yang J, Hou Y, et al. Casticin inhibits nasopharyngeal carcinoma growth by targeting phosphoinositide 3-kinase[J]. Cancer cell international, 2019, 19(1): 348.
[6] Yang F, He K, Huang L, et al. Casticin inhibits the activity of transcription factor Sp1 and the methylation of RECK in MGC803 gastric cancer cells[J]. Experimental and Therapeutic Medicine,

2017, 13(2): 745-750.
[7] Zhou L, Dong X, Wang L, et al. Casticin attenuates liver fibrosis and hepatic stellate cell activation by blocking TGF-β/Smad signaling pathway[J]. Oncotarget, 2017, 8(34): 56267.
[8] Shang H S, Chen K W, Chou J S, et al. Casticin inhibits in vivo growth of xenograft tumors of human oral cancer SCC-4 cells[J]. in vivo, 2020, 34(5): 2461-2467.

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