Casticin

Cat. No.: HY-N0516

CAS No.: 479-91-4

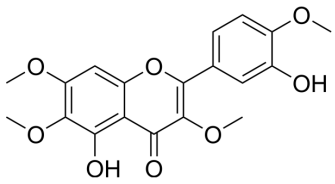
Molecular Formula: $C_{19}H_{18}O_8$

Molecular Weight: 374.34

Target: STAT

Pathway: JAK/STAT Signaling; Stem Cell/Wnt

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (267.14 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6714 mL	13.3568 mL	26.7137 mL
	5 mM	0.5343 mL	2.6714 mL	5.3427 mL
	10 mM	0.2671 mL	1.3357 mL	2.6714 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 6.25 mg/mL (16.70 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Casticin is a methyoxylated flavonol isolated from Vitex rotundifolia, with antimitotic and anti-inflammatory effect. Casticin inhibits the activation of STAT3.

IC_{50} & Target

STAT3[3]

In Vitro

Casticin (0.2-1.0 μM) dose-dependently inhibits the proliferation of KB cells, with an IC_{50} of 0.23 μM on day 3, while shows no significant inhibition on 3T3 Swiss Albino and TIG-103 cells. Casticin (0.6 μM) alters spindle morphology with partial mitotic spindle breakdown or with disordered spindles[1]. Casticin (0-40 μM) dose-dependently inhibits the proliferation of LX2 cells. Casticin (40 μM) suppresses L02 cells proliferation and induces apoptosis. Casticin inhibits fibrotic effects of TGF-β1 on ECM deposition in LX2 cells by evaluating the mRNA levels of TGF-β, collagen α1(I), MMP-2, MMP-9, TIMP-1 and TIMP-2[2]. Casticin (0-8 μM) reduces the viability of 786-O, YD-8, and HN-9 cells, but shows no significant effect on that of the normal HEL 299 cells. Casticin (5 μM) increases cleavage caspase-3 and PPAR, diminishes the levels of B-cell lymphoma-extra large (Bcl-xl), Bcl-2, IAP-1/-2, vascular endothelial growth factor (VEGF), matrix metallopeptidase 9 (MMP-9), and cyclooxygenase 2 (COX-2) proteins in 786-O, YD-8, and HN-9 cells. Casticin (5 μM) also promotes apoptotic cell death, inhibits constitutively active

	STAT3 in tumor cells, modulates STAT3 activation by altering the activity of upstream STAT3 regulators, and abrogates IL-6-induced STAT3 activation. In addition, Casticin (2.5 μM) enhances the effect of ionizing radiation in 786-O cells and potentiates the therapeutic effect of radiotherapy[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Casticin (20 mg/kg, p.o.) has toxic effect on the liver in mice with CCl4-and BDL-induced hepatic injury. Casticin attenuates liver fibrosis induced by CCl4 or BDL in vivo. Casticin inhibits HSC activation and collagen matrix expression by blocking TGF-β/Smad signaling in vivo[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	LX-2 cells or L02 cells are plated at a density of 5×10^3 cells per well in a 96-well plate and treated with Casticin (0-40 μM) for 48 h in growth medium containing serum. Cell proliferation is determined using a CCK-8 assay kit[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] Male mice 6-8 weeks of age weighing 20-30 g are kept in a temperature-controlled room with an alternating 12 h dark and light cycle. A total of 32 mice are divided randomly into four groups of 8 animals each: control, Casticin, CCl4, and CCl4 + Casticin. To induce liver fibrosis, CCl4 dissolved in olive oil (20%) is injected intraperitoneally into mice (1.0 mL/kg body weight) in the CCl4 and CCl4 + Casticin groups twice a week for six weeks. Mice in the control group and Casticin group are injected with an equivalent volume of olive oil. Casticin is dissolved in 0.25% Tween-80. After treatment with CCl4 or olive oil for six weeks, mice in the Casticin group and CCl4 + Casticin group receives Casticin (20 mg/kg) by gastric gavage daily for two weeks, and the other two groups are given the equivalent volume of 0.25% Tween-80. After the eight week intervention period, mice are euthanatized under 3% pentobarbital sodium anesthesia (40 mg/kg ip), and the livers and blood from all animals are collected. Serum is obtained by centrifugation (1600 g, 15 min) and stored at −20°C for further examination[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Genes (Basel). 2022 May 3;13(5):815.

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REFERENCES

[1]. Kobayakawa J, et al. G2-M arrest and antimitotic activity mediated by casticin, a flavonoid isolated from Viticis Fructus (Vitex rotundifolia Linne fil.). Cancer Lett. 2004 May 10;208(1):59-64.

[2]. Zhou L, et al. Casticin attenuates liver fibrosis and hepatic stellate cell activation by blocking TGF-β/Smad signaling pathway. Oncotarget. 2017 Apr 27;8(34):56267-56280.

[3]. Lee JH, et al. Casticin inhibits growth and enhances ionizing radiation-induced apoptosis through the suppression of STAT3 signaling cascade. J Cell Biochem. 2018 Dec 5.

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

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