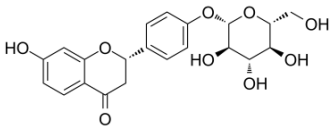
Liquiritin

Cat. No.:	HY-N0376
CAS No.:	551-15-5
Molecular Formula:	$C_{21}H_{22}O_9$
Molecular Weight:	418.39
Target:	Reactive Oxygen Species
Pathway:	Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 150 mg/mL (**358.52 mM;** Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.3901 mL	11.9506 mL	23.9011 mL
5 mM	0.4780 mL	2.3901 mL	4.7802 mL
10 mM	0.2390 mL	1.1951 mL	2.3901 mL

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

In Vivo

1. Add each solvent one by one: **10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline**
   Solubility: ≥ 2.5 mg/mL (5.98 mM); Clear solution
2. Add each solvent one by one: **10% DMSO >> 90% (20% SBE-β-CD in saline)**
   Solubility: ≥ 2.5 mg/mL (5.98 mM); Clear solution
3. Add each solvent one by one: **10% DMSO >> 90% corn oil**
   Solubility: ≥ 2.5 mg/mL (5.98 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Liquiritin, a flavonoid isolated from Glycyrrhiza, is a potent and competitive **AKR1C1** inhibitor with **IC_{50}**s of 0.62 μM, 0.61 μM, and 3.72μM for AKR1C1, AKR1C2 and AKR1C3, respectively. Liquiritin efficiently inhibits progesterone metabolism mediated by AKR1C1 in vivo[1]. Liquiritin acts as an antioxidant and has neuroprotective, anti-cancer and anti-inflammatory activity[2].

IC_{50} & Target

IC50: 0.62 μM (AKR1C1), 0.61 μM (AKR1C2) and 3.72μM (AKR1C3)[1]

In Vitro	Liquiritin can target the residues Ala-27, Val-29, Ala-25, and Asn-56 of AKR1C1[1]. Liquiritin (50 μM; 6 hours) results in 85.00% of reduction in progesterone metabolism, which is mediated by Aldo-keto reductase family 1 member C1 (AKR1C1) enzymatic activity in HEC-1-B cells[1]. Liquiritin (100 μM) increases glucose-6-phosphate dehydrogenase expression on B65 neuroblastoma cells[2].

REFERENCES

[1]. Nakatani Y, et al. Neuroprotective effect of liquiritin as an antioxidant via an increase in glucose-6-phosphate dehydrogenase expression on B65 neuroblastoma cells. Eur J Pharmacol. 2017 Nov 15;815:381-390.

[2]. Zeng C, et al. Liquiritin, as a Natural Inhibitor of AKR1C1, Could Interfere With the Progesterone Metabolism. Front Physiol. 2019 Jul 3;10:833.

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