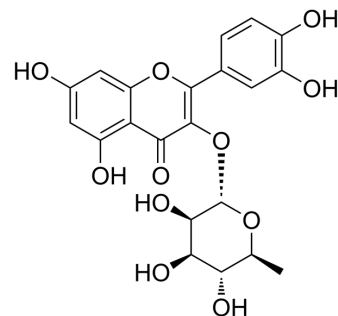


Quercitrin

Cat. No.:	HY-N0418
CAS No.:	522-12-3
Molecular Formula:	C ₂₁ H ₂₀ O ₁₁
Molecular Weight:	448.38
Target:	Ribosomal S6 Kinase (RSK); Autophagy; Reactive Oxygen Species; Apoptosis
Pathway:	MAPK/ERK Pathway; Autophagy; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB; Apoptosis
Storage:	-20°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro	DMSO : 125 mg/mL (278.78 mM; Need ultrasonic)					
	Preparing Stock Solutions	Solvent	Mass	1 mg	5 mg	10 mg
		Concentration				
		1 mM		2.2303 mL	11.1513 mL	22.3025 mL
		5 mM		0.4461 mL	2.2303 mL	4.4605 mL
	10 mM		0.2230 mL	1.1151 mL	2.2303 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: ≥ 2.58 mg/mL (5.75 mM); Clear solution					
	2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.08 mg/mL (4.64 mM); Clear solution					

BIOLOGICAL ACTIVITY

Description	Quercitrin (Quercetin 3-rhamnoside) is a bioflavonoid compound with potential anti-inflammation, antioxidative and neuroprotective effect. Quercitrin induces apoptosis of colon cancer cells. Quercitrin can be used for the research of cardiovascular and neurological disease research ^{[1][2]} .
In Vitro	<p>Quercitrin (5-50 μM; 24-72 h) time- and dose-dependently inhibits cell proliferation and increases cytotoxic effects to colorectal carcinoma cells^[1].</p> <p>Quercitrin (5-50 μM; 24-72 h) time- and dose-dependently increases nucleosomal enrichment factor (EF) of DLD-1 cells^[1].</p> <p>Quercitrin (50 μM; 48-72 h) induces cell apoptosis and the loss of mitochondrial membrane potential, and causes translocation of phosphatidylserine (PS) from the inner to outer Leaflet of DLD-1 cells^[1].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Cell Proliferation Assay^[1]</p>

	<table border="1"> <tr> <td>Cell Line:</td> <td>DLD-1 colon cancer cell lines</td> </tr> <tr> <td>Concentration:</td> <td>5, 10, 25 and 50 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>24, 48 and 72 hours</td> </tr> <tr> <td>Result:</td> <td>Time- and Dose-dependently decreased cell proliferation of colorectal carcinoma cells.</td> </tr> </table> <p>Apoptosis Analysis^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>DLD-1 colon cancer cell lines</td> </tr> <tr> <td>Concentration:</td> <td>50 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>48 and 72 hours</td> </tr> <tr> <td>Result:</td> <td>Induced cell apoptosis and time- and dose-dependently increased caspase-3 enzyme activity.</td> </tr> </table>	Cell Line:	DLD-1 colon cancer cell lines	Concentration:	5, 10, 25 and 50 μ M	Incubation Time:	24, 48 and 72 hours	Result:	Time- and Dose-dependently decreased cell proliferation of colorectal carcinoma cells.	Cell Line:	DLD-1 colon cancer cell lines	Concentration:	50 μ M	Incubation Time:	48 and 72 hours	Result:	Induced cell apoptosis and time- and dose-dependently increased caspase-3 enzyme activity.
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In Vivo	<p>Quercitrin (50 and 100 mg/kg; oral gavage, once) shows effective protection against brain injury in mice by inhibiting oxidative stress and inflammation induced by carbon tetrachloride^[2].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1"> <tr> <td>Animal Model:</td> <td>Male ICR mice with carbon tetrachloride (CCl₄) induced brain injury^[2]</td> </tr> <tr> <td>Dosage:</td> <td>50 and 100 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>Oral gavage; 50 and 100 mg/kg, once</td> </tr> <tr> <td>Result:</td> <td>Dose-dependently decreased the levels of ROS and malondialdehyde (MDA) concentration in the hippocampus homogenates, and also dose-dependently decreased the CYP2E1 level in the brains. Increased the activities of superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx). Inhibited the N-methyl-D-aspartate receptor 2B subunit (NR2B) level and the activities of monoamine oxidase (MAO) and acetylcholine esterase (AChE) in mouse brains.</td> </tr> </table>	Animal Model:	Male ICR mice with carbon tetrachloride (CCl ₄) induced brain injury ^[2]	Dosage:	50 and 100 mg/kg	Administration:	Oral gavage; 50 and 100 mg/kg, once	Result:	Dose-dependently decreased the levels of ROS and malondialdehyde (MDA) concentration in the hippocampus homogenates, and also dose-dependently decreased the CYP2E1 level in the brains. Increased the activities of superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx). Inhibited the N-methyl-D-aspartate receptor 2B subunit (NR2B) level and the activities of monoamine oxidase (MAO) and acetylcholine esterase (AChE) in mouse brains.								
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CUSTOMER VALIDATION

- Acta Pharm Sin B. 2021 Jan;11(1):143-155.

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REFERENCES

- [1]. Cincin ZB, et al. Apoptotic Effects of Quercitrin on DLD-1 Colon Cancer Cell Line. Pathol Oncol Res. 2015 Apr;21(2):333-8.
- [2]. Cincin ZB, et al. Apoptotic Effects of Quercitrin on DLD-1 Colon Cancer Cell Line. Pathol Oncol Res. 2015 Apr;21(2):333-8.

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

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