Schisandrol B

Cat. No.: HY-N0692 CAS No.: 58546-54-6 Molecular Formula: C₂₃H₂₈O₇ Molecular Weight: 416.46

Autophagy; Reactive Oxygen Species; Cytochrome P450 Target:

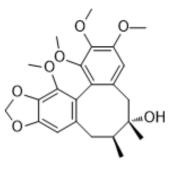
Pathway: Autophagy; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κΒ

Storage: Powder -20°C 3 years

4°C 2 years

In solvent -80°C 2 years

> -20°C 1 year



Product Data Sheet

SOLVENT & SOLUBILITY

In Vitro

DMSO: 50 mg/mL (120.06 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent Mass Concentration	1 mg	5 mg	10 mg
	1 mM	2.4012 mL	12.0060 mL	24.0119 mL
	5 mM	0.4802 mL	2.4012 mL	4.8024 mL
	10 mM	0.2401 mL	1.2006 mL	2.4012 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 (6.00 mM); Clear solution
- 2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: 2.5 mg/mL (6.00 mM); Suspended solution; Need ultrasonic
- 3. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (6.00 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	Schisandrol B (Gomisin-A) is a major active constituent of Schisandra chinensis with hepato-protective effects. Schisandrol B inhibits reactive oxygen species (ROS) production. Schisandrol B inhibits the activity of P-glycoprotein and CYP3A and also has anti-inflammatory, anti-diabetic and antioxidant activities ^{[1][2][3]} .		
IC ₅₀ & Target	CYP2	СҮРЗА	
In Vitro	Schisandrol B (Gomisin-A; 1-10 μM; 2 days) treatment decreases the aging related inflammatory molecules, such as, COX-2,		

IL1 β , and TNF- α . Schisandrol B attenuates the activity of senescence-associated β -galactosidase^[2].

Schisandrol B (Gomisin-A; 1-10 μ M; 2 days) inhibits reactive oxygen species production even in the stress-induced premature senescence (SIPS)-human diploid fibroblast (HDF) cells^[2].

Schisandrol B (Gomisin-A; 1-10 μM) inhibits the MAPK pathway and the translocation of NF-κB to the nucleus^[2].

Schisandrol B (Gomisin-A; 1-10 μ M) promotes the autophagy and mitochondrial biogenesis factors through the translocation of Nrf-2, and inhibits aging progression in the SIPS-HDF cells^[2].

Schisandrol B (0-80 μ M) dramatically alters APAP metabolic activation by inhibiting the activities of CYP2E1 and CYP3A11^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis^[2]

Cell Line:	Human diploid fibroblast (HDF) cells	
Concentration:	1 μΜ, 10 μΜ	
Incubation Time:	3 days	
Result:	Decreased the aging related inflammatory molecules, such as, COX-2, IL1 β , and TNF- α .	

In Vivo

Schisandrol B (12.5 -200 mg/kg; oral administration; seven times with an interval of 12 hours) pretreatment significantly attenuates the increases in alanine aminotransferase and aspartate aminotransferase activity, and prevents elevated hepatic malondialdehyde formation and the depletion of GSH in a dose-dependent manner. Schisandrol B abrogates APAP-induced activation of p53 and p21, and increases expression of liver regeneration and antiapoptotic-related proteins such as cyclin D1 (CCND1), PCNA, and BCL-2^[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male C57BL/6 mice (6-8 weeks old, 20-22 g) injected with Acetaminophen (APAP) ^[1]	
Dosage:	12.5 mg/kg, 12.5 mg/kg and 200 mg/kg	
Administration:	Oral administration; seven times with an interval of 12 hours	
Result:	Showed a protective effect against APAP-induced liver injury in mice.	

CUSTOMER VALIDATION

• Front Cell Dev Biol. 2021 Nov 10;9:763864.

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REFERENCES

[1]. Jiang Y, et al. Schisandrol B protects against acetaminophen-induced hepatotoxicity by inhibition of CYP-mediated bioactivation and regulation of liver regeneration. Toxicol Sci. 2015 Jan;143(1):107-15.

[2]. Jin J, et al. Enhancement of oral bioavailability of paclitaxel after oral administration of Schisandrol B in rats. Biopharm Drug Dispos. 2010 May;31(4):264-8.

[3]. Jeong-Seok Kim, et al. Gomisin A modulates aging progress via mitochondrial biogenesis in human diploid fibroblast cells. Clin Exp Pharmacol Physiol. 2018 Jun;45(6):547-555.

 $\label{lem:caution:Product} \textbf{Caution: Product has not been fully validated for medical applications. For research use only.}$

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