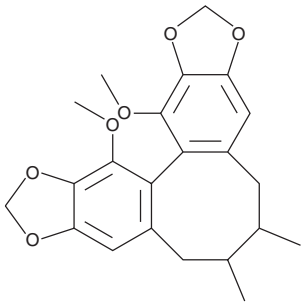
PRODUCT INFORMATION

Schisandrin C

Item No. 27475

CAS Registry No.:	61301-33-5
Formal Name:	(6R,7S,13aS)-5,6,7,8-tetrahydro-13,14-dimethoxy-6,7-dimethyl-cycloocta[1,2-f:3,4-f']*bis*[1,3]benzodioxole
Synonyms:	Schizandrin C, Wuweizisu C
MF:	$C_{22}H_{24}O_6$
FW:	384.4
Purity:	≥98%
UV/Vis.:	λ_{max}: 220 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years
Item Origin:	Plant/*Schisandra chinensis*



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Schisandrin C is supplied as a crystalline solid. A stock solution may be made by dissolving the schisandrin C in the solvent of choice, which should be purged with an inert gas. Schisandrin C is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of schisandrin C in these solvents is approximately 30 mg/ml.

Description

Schisandrin C is a lignan originally isolated from *Schizandrae* that has diverse biological activities.[1-5] It decreases viability of U937 cells in a concentration-dependent manner and induces cell cycle arrest at the G_1 phase when used at a concentration of 100 µM.[2] Schisandrin C (5-20 µM) decreases hydrogen peroxide-induced cell death and production of reactive oxygen species (ROS) in C2C12 skeletal muscle cells.[3] It also decreases levels of matrix metalloproteinase-2 (MMP-2), MMP-9, COX-2, VCAM-1, IL-1β, and TNF-α in hydrogen peroxide-stimulated C2C12 cells. Schisandrin C decreases lipoteichoic acid-induced production of nitric oxide (NO), prostaglandin E_2 (PGE_2; Item No. 14010), TNF-α, IL-1β, and IL-6 in mouse primary microglia.[4] *In vivo*, schisandrin C (200 mg/kg) decreases serum alanine amino transferase (ALT) and aspartate amino transferase (AST) activity, increases hepatic mitochondrial and total glutathione (GSH) levels, and reduces liver injury in a mouse model of acetaminophen-induced liver injury partially *via* inhibition of the cytochrome P450 (CYP) isoforms CYP2E1, CYP1A2, and CYP3A11.[5]

References

1. Li, X.Y. *Mem. Inst. Oswaldo Cruz.* **86(Suppl 2)**, 31-37 (1991).
2. Park, C., Choi, Y.-W., Hyun, S.K., *et al. Int. J. Mol. Med.* **24(4)**, 495-502 (2009).
3. Kim, J.S. and Yi, H.K. *Naunyn Schmiedebergs Arch. Pharmacol.* **391(2),** 197-206 (2018).
4. Park, S.Y., Park, S.J., Park, T.G., *et al. Int. Immunopharmacol.* **17(2),** 415-426 (2013).
5. Jiang, Y., Fan, X., Wang, Y., *et al. Chem. Biol. Interact.* **231**, 83-89 (2015).

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