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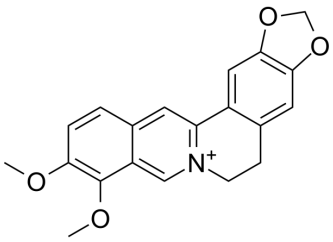
Berberine

Cat. No.:	HY-N0716
CAS No.:	2086-83-1
Molecular Formula:	$C_{20}H_{18}NO_4^+$
Molecular Weight:	336.36
Target:	Topoisomerase; Autophagy; Bacterial; Reactive Oxygen Species; Antibiotic; Parasite; Apoptosis; PI3K; Akt; Caspase; JNK; AP-1; NF-κB
Pathway:	Cell Cycle/DNA Damage; Autophagy; Anti-infection; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB; Apoptosis; PI3K/Akt/mTOR; MAPK/ERK Pathway
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Berberine (Natural Yellow 18) is an alkaloid isolated from the Chinese herbal medicine Huanglian, as an antibiotic. Berberine (Natural Yellow 18) induces reactive oxygen species (ROS) generation and inhibits DNA topoisomerase. Berberine (Natural Yellow 18) has antineoplastic properties. The sulfate form (HY-N0716B) improves bioavailability[1].
IC_{50} & Target	ROS[1] DNA topoisomerase[1]
In Vitro	Berberine (1.25-160 μM; 72 hours) has potential inhibitory effects on the proliferation of four colorectal carcinoma cell lines LoVo, HCT116, SW480, and HT-29[1]. Berberine (1.25-160 μM; 24-72 hours) induces a time- and dose-dependent inhibition of LoVo cell growth[1]. LoVo cells are exposure to Berberine (10-80 μM) for 24 h. Cell cycle analysis of 40 μM Berberine-treated LoVo cells by flow cytometry shows accumulation of cells in the G2/M phase[1]. Berberine (10-80 μM) suppresses cyclin B1, cdc2 and cdc25c protein expression after 24 h, especially at the dose of 80.0 μM[1]. Berberine exhibits antimicrobial acitivity through inhibition of cell division protein FtsZ, or through DNA/RNA binding and deal thus DNA/RNAdamege[3]. Berberine exhibts anti-inflammatory activity by inhibiting TNF-α and the activation of its downstream pathway AP-1 and NF-kB[4]. Berberine exhibits neuroprotective efficacy by inhibiting the reactive oxygen species (ROS) production and caspase activation, and activatiing the PI3K/Akt signaling pathway, and heme oxygenase-1 (HO-1) expression[5]. Berberine attenuates metabolic diseases through regulations of the lipids composition and inhibition of insulin resistance[6]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	Four colorectal carcinoma cell lines LoVo, HCT116, SW480, and HT-29
Concentration:	1.25, 2.5, 5, 10, 20, 40, 80, and 160 μM
Incubation Time:	72 hours
Result:	Inhibited the proliferation of four cell lines. The IC_{50} ranged from 40.8±4.1 μM (LoVo) to

	98.6±2.9 μM (HCT116).

Cell Proliferation Assay[1]

Cell Line:	Colorectal carcinoma cell lines LoVo
Concentration:	1.25, 2.5, 5, 10, 20, 40, 80, and 160 μM
Incubation Time:	24, 48, 72 hours
Result:	Induced a time- and dose-dependent inhibition of cell growth. By 72 h, 160.0 μM induced 71.1±1.9 % growth inhibitions in LoVo cells.

Cell Cycle Analysis[1]

Cell Line:	LoVo cells
Concentration:	0, 10, 20, 40, or 80 μM
Incubation Time:	24 hours
Result:	Exposure to 40.0 μM induced G2/M-phase cell cycle arrest, an increase in the G2/M-phase population and a progressive decline in the G1 population.

Western Blot Analysis[1]

Cell Line:	LoVo cells
Concentration:	10, 20, 40, or 80 μM
Incubation Time:	24 hours
Result:	Suppressed cyclin B1, cdc2 and cdc25c protein expression.

In Vivo

Berberine (10, 30, or 50 mg/kg/day; gastrointestinal gavage; for 10 consecutive days) inhibits the growth of human colorectal adenocarcinoma in vivo. Berberine at doses of 30 and 50 mg/kg/day taken by gastrointestinal gavage shows inhibitory rates of 33.1% and 45.3% on the human colorectal adenocarcinoma xenograft growth in nude mice[1].

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

Animal Model:	5-week-old BALB/c nu/nu mice with human colorectal adenocarcinoma LoVo xenografts[1]
Dosage:	10, 30, or 50 mg/kg/day
Administration:	Gastrointestinal gavage; for 10 consecutive days
Result:	Showed inhibitory rates of 33.1 % and 45.3 % at doses of 30 and 50 mg/kg/day.

CUSTOMER VALIDATION

- Acta Pharmacol Sin. 2022 Aug 10.
- Int J Nanomedicine. 2023 Jul 31.
- JCI Insight. 2023 Jul 24;8(14):e166306.
- Phytomedicine. 2023 Dec 2, 155247.

- Phytomedicine. 2023 Jul 17, 154962.

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REFERENCES

[1]. Cui HX, et al. Preparation and Evaluation of Antidiabetic Agents of Berberine Organic Acid Salts for Enhancing the Bioavailability. Molecules. 2018 Dec 28;24(1):103.

[2]. Boberek JM, et al., Genetic evidence for inhibition of bacterial division protein FtsZ by berberine. PLoS One. 2010 Oct 29;5(10):e13745.

[3]. Remppis A, et al., Rhizoma Coptidis inhibits LPS-induced MCP-1/CCL2 production in murine macrophages via an AP-1 and NFkappaB-dependent pathway. Mediators Inflamm. 2010;2010:194896.

[4]. Bae J, et al., Berberine protects 6-hydroxydopamine-induced human dopaminergic neuronal cell death through the induction of heme oxygenase-1. Mol Cells. 2013 Feb;35(2):151-7.

[5]. Ye Y, et al., Efficacy and Safety of Berberine Alone for Several Metabolic Disorders: A Systematic Review and Meta-Analysis of Randomized Clinical Trials. Front Pharmacol. 2021 Apr 26;12:653887.

[6]. Cai Y, et al. Berberine inhibits the growth of human colorectal adenocarcinoma in vitro and in vivo. J Nat Med. 2014 Jan;68(1):53-62.

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

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