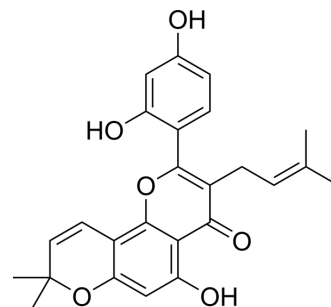


Morusin

Cat. No.:	HY-N0622		
CAS No.:	62596-29-6		
Molecular Formula:	C ₂₅ H ₂₄ O ₆		
Molecular Weight:	420.45		
Target:	NF-κB; STAT; Bacterial; NF-κB		
Pathway:	NF-κB; JAK/STAT Signaling; Stem Cell/Wnt; Anti-infection		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (237.84 mM)
 * "≥" means soluble, but saturation unknown.

	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM	2.3784 mL	11.8920 mL	23.7840 mL
	5 mM	0.4757 mL	2.3784 mL	4.7568 mL
	10 mM	0.2378 mL	1.1892 mL	2.3784 mL

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

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
 Solubility: ≥ 2.5 mg/mL (5.95 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
 Solubility: ≥ 2.5 mg/mL (5.95 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	Morusin is a prenylated flavonoid isolated from <i>Morus alba</i> Linn. with various biological activities, such as antitumor, antioxidant, and anti-bacteria property. Morusin could inhibit NF-κB and STAT3 activity.	
IC₅₀ & Target	p65	STAT3
In Vitro	Morusin exhibits a dose- and time-dependent inhibitory effect on murine and human breast cancer cells. IC ₅₀ is 9.48 μg/mL for normal mammary epithelial cells (MCF-10A); 2.03 and 1.87 μg/mL for murine breast cancer cells (4 T1 and EMT6); and 2.71 and 3.86 μg/mL for human breast cancer cells (MCF-7 and MDA-MB-231), respectively, the maximal inhibition of cell growth (>80 %) is obtained at 8 μg/mL. The apoptotic cells in morusin treated breast cancer cells are increased significantly	

in a dose-dependent manner^[1]. Morusin significantly inhibits the growth and clonogenicity of human colorectal cancer HT-29 cells. Morusin also inhibits the phosphorylation of IKK- α , IKK- β and I κ B- β , increases expression of I κ B- α , and suppresses nuclear translocation of NF- κ B and its DNA binding activity. Dephosphorylation of NF- κ B upstream regulators PI3K, Akt and PDK1 is also displayed. In addition, activation of caspase-8, change of mitochondrial membrane potential, release of cytochrome c and Smac/DIABLO, and activation of caspase-9 and -3 are observed at the early time point. Downregulation in the expression of Ku70 and XIAP is exhibited afterward^[2]. Morusin suppresses viability of prostate cancer cells, but little effect in normal human prostate epithelial cells. Morusin also reduces STAT3 activity by inhibiting its phosphorylation, nuclear accumulation, and DNA binding activity. In addition, morusin down-regulated expression of STAT3 target genes encoding Bcl-xL, Bcl-2, Survivin, c-Myc and Cyclin D1. It induces apoptosis in human prostate cancer cells by reducing STAT3 activity^[3].

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

In Vivo

Morusin retards the growth of breast cancer significantly. Mean tumor weight of the control mice is 1.14 \pm 0.30 g, and those of the mice administrated with 5 and 10 mg/kg of morusin are 0.61 \pm 0.23 and 0.41 \pm 0.10 g, respectively, tumor inhibitory rates are 46.5 %, and 64.1 %, respectively^[1].

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

PROTOCOL

Cell Assay ^[1]

The cytotoxicity of morusin against human normal mammary epithelial cells and murine breast cancer cells (4 T1 and EMT6) and human breast cancer cells (MCF-7 and MDA-MB-231) is tested by modified MTT assay [23]. Cells are treated with various concentrations of morusin (1, 2, 4, 6 and 8 μ g/mL). After treatment with morusin for 1, 2, 3, 4, and 5 days, 20 μ L MTT (pH 4.7) is added to each well, and cultivated for another 4 h, 100 μ L of 10 % SDS/0.01 N HCl is added and incubated at 37 $^{\circ}$ C overnight to dissolve the formazan. Absorbance is measured at 570 nm^[1].

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

Animal Administration ^[1]

Mice: Two treatment group mice are injected with 5 and 10 mg/kg of morusin i.p. three times weekly for 4 weeks, respectively, and the control mice are injected with DMSO. During the experiment, mice are weighted, and tumor volumes are measured weekly using calipers and their volumes are calculated^[1].

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

CUSTOMER VALIDATION

- Stem Cell Res Ther. 2021 Mar 12;12(1):173.
- Drug Des Devel Ther. 2020 Mar 26;14:1227-1240.
- Biochem Biophys Res Commun. 2018 Sep 3;503(1):297-303.
- Ann Transl Med. 2020 Mar;8(6):327.

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REFERENCES

[1]. Li H, et al. Morusin suppresses breast cancer cell growth in vitro and in vivo through C/EBP β and PPAR γ mediated lipooapoptosis. J Exp Clin Cancer Res. 2015 Nov 4;34:137.

[2]. Lee JC, et al. Morusin induces apoptosis and suppresses NF-kappaB activity in human colorectal cancer HT-29 cells. Biochem Biophys Res Commun. 2008 Jul 18;372(1):236-42.

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

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