

Data Sheet

Product Name: Morusin

Cat. No.: CS-6885

CAS No.: 62596-29-6

Molecular Formula: C25H24O6

Molecular Weight: 420.45

Target: Bacterial; NF-κΒ; STAT

Pathway: Anti-infection; JAK/STAT Signaling; NF-κΒ; Stem Cell/Wnt

Solubility: DMSO : \geq 125 mg/mL (297.30 mM)

BIOLOGICAL ACTIVITY:

Morusin is a prenylated flavonoid isolated from M. australis with various biological activities, such as antitumor, antioxidant, and antibacteria property. Morusin could inhibit NF-kB and STAT3 activity. IC50 & Target: NF-kB^[1] In Vitro: Morusin exhibits a dose- and time-dependent inhibitory effect on murine and human breast cancer cells. IC₅₀ is 9.48 μ g/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-βand IκΒ-β, increases expression of IκB-α, and suppresses nuclear translocation of NF-κB 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]. In Vivo: Morusin retards the growth of breast cancer significantly. Mean tumor weight of the control mice is 1.14±0.30 g, and those of the mice administrated with 5 and 10 mg/kg of morusin are 0.61±0.23 and 0.41±0.10 g, respectively, tumor inhibitory rates are 46.5 %, and 64.1 %, respectively^[1].

PROTOCOL (Extracted from published papers and Only for reference)

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 °C overnight to dissolve the formazan. Absorbance is measured at 570 nm $^{[1]}$. Animal Administration: Morusin is prepared in DMSO. $^{[1]}$ 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 $^{[1]}$.

References:

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[1]. Li H, et al. Morusin suppresses breast cancer cell growth in vitro and in vivo through C/EBPβ and PPARγ mediated lipoapoptosis. 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.

[3]. Lim SL, et al. Morusin induces cell death through inactivating STAT3 signaling in prostate cancer cells. Am J Cancer Res. 2014 Dec 15;5(1):289-99.

CAIndexNames:

4H,8H-Benzo[1,2-b:3,4-b'] dipyran-4-one, 2-(2,4-dihydroxyphenyl)-5-hydroxy-8,8-dimethyl-3-(3-methyl-2-buten-1-yl)-1-hydroxy-8,8-dimethyl-3-(3-methyl-2-buten-1-yl)-1-hydroxy-8,8-dimethyl-3-(3-methyl-2-buten-1-yl)-1-hydroxy-8,8-dimethyl-3-(3-methyl-2-buten-1-yl)-1-hydroxy-8,8-dimethyl-3-(3-methyl-2-buten-1-yl)-1-hydroxy-8,8-dimethyl-3-(3-methyl-2-buten-1-yl)-1-hydroxy-8,8-dimethyl-3-(3-methyl-3-buten-1-yl)-1-hydroxy-8,9-dimethyl-3-(3-methyl-3-buten-1-yl)-1-hydroxy-8,9-dimethyl-3-(3-methyl-3-buten-1-yl)-1-hydroxy-8,9-dimethyl-3-(3-methyl-3-buten-1-yl)-1-hydroxy-8,9-dimethyl-3-(3-methyl-3-buten-1-yl)-1-hydroxy-8,9-dimethyl-3-(3-methyl-3-buten-1-yl)-1-hydroxy-8,9-dimethyl-3-(3-methyl-3-buten-1-yl)-1-hydroxy-8,9-dimethyl-3-(3-methyl-3-buten-1-yl)-1-hydroxy-8,9-dimethyl-3-(3-methyl-3-buten-1-yl)-1-hydroxy-8,9-dimethyl-3-(3-methyl-3-buten-1-yl)-1-hydroxy-9,9-dimet

SMILES:

 $O = C1C2 = C(O)C = C3C(C = CC(C)(C)O3) = C2OC(C4 = CC = C(O)C = C4O) = C1C/C = C(C) \setminus C$

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

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