Bigelovin

Cat. No.: HY-116506

CAS No.: 3668-14-2

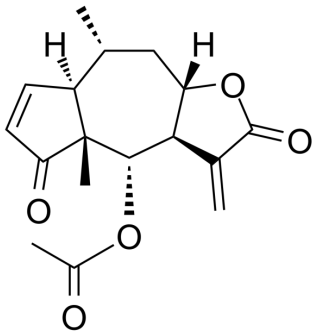
Molecular Formula: $C_{17}H_{20}O_5$

Molecular Weight: 304.34

Target: RAR/RXR; Reactive Oxygen Species; Apoptosis; Autophagy

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

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (328.58 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.2858 mL	16.4290 mL	32.8580 mL
	5 mM	0.6572 mL	3.2858 mL	6.5716 mL
	10 mM	0.3286 mL	1.6429 mL	3.2858 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 (8.21 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.21 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.21 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Bigelovin, a sesquiterpene lactone isolated from Inula helianthus-aquatica, is a selective retinoid X receptor α agonist. Bigelovin suppresses tumor growth through inducing apoptosis and autophagy via the inhibition of mTOR pathway regulated by ROS generation[1].

In Vitro

Bigelovin (0-20 μM, 24-72 h) significantly inhibits cell viability of liver cancer cells and induces apoptosis and autophagy[1].
Bigelovin causes a significant increase of p62, LC3B-II, Beclin-1 and a corresponding decrease of p62 levels in a time-dependent manner[1].
Bigelovin induces cell death involves the suppression of mTOR pathway regulated by ROS production[1].

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

Cell Viability Assay[1]

Cell Line:	HepG2 and SMMC-7721 cells.
Concentration:	0-20 μM.
Incubation Time:	24, 48, 72 h.
Result:	Significantly reduced the cell viability of HepG2 and SMMC-7721 cells in a dose- and timedependent manner. No significant difference observed in cell viability of normal liver cell lines, LO2 andLX2, after BigV treatment for 24, 48 or 72 h.

Western Blot Analysis[1]

Cell Line:	HepG2 and SMMC-7721 cells.
Concentration:	0-10 μM.
Incubation Time:	24 h.
Result:	The expression of Bcl-2 was decreased, whereas Bax was increased after treatment with BigV. Moreover, Caspase-9, -3 and PARP cleavage were activated significantly after BigV treatment.

In Vivo

Bigelovin (BigV, 5, 10, 20 mg/kg) exerts anti-tumor activity in HepG2 xenograft tumor model[1].

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Animal Model:	HepG2 xenograft model based on the male athymic BALB/c nude mice (5-6 weeks old, 18-22 g)[1].
Dosage:	5, 10, 20 mg/kg.
Administration:	Intravenous injection every 2 days.
Result:	The tumor growth rate was significantly slower in BigV treated groups in a dose-dependent manner, along with the reduced tumor weight. No significant alteration of body weight and hepatic enzyme levels (AST, ALT and LDH) in serum was observed after BigV administration. Western blot findings of tumor tissues indicated the activation of apoptosis and autophagy characterized by the increase of cleaved Caspase-3 and PARP, as well as LC3BIIlevels. The inactivation of mTOR was also observed in tumor tissuesisolated from BigV-treated mice.

REFERENCES

[1]. Bei Wang, et al. Bigelovin, a sesquiterpene lactone, suppresses tumor growth through inducing apoptosis and autophagy via the inhibition of mTOR pathway regulated by ROS generation in liver cancer. Biochem Biophys Res Commun. 2018 May 5;499(2):156-163.

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

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