Ascochlorin

Cat. No.: HY-101021

CAS No.: 26166-39-2

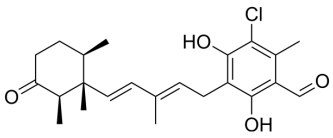
Molecular Formula: $C_{23}H_{29}ClO_4$

Molecular Weight: 404.93

Target: STAT; Apoptosis; Antibiotic

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

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Ascochlorin (Ilicicolin D), an isoprenoid antibiotic, mediates its anti-tumor effects predominantly through the suppression of STAT3 signaling cascade. Ascochlorin induces apoptosis. Anti-inflammatory activity[1][2][3].

IC_{50} & Target	STAT3	Apoptosis

In Vitro

Ascochlorin (Ilicicolin D) (10-50 μM; 24-72 hours) inhibits the viability of HepG2, HCCLM3 and Huh7 cells in a time and dose dependent manner[3].

Ascochlorin (50 μM; 48 hours) induces apoptosis in HCC cells[3].

Ascochlorin (1-50 μM) significantly suppresses the production of nitric oxide (NO) and prostaglandin E2 (PGE2) and decreases the gene expression of inducible NO synthase (iNOS) and cyclooxygenase-2 (COX-2) in a dose-dependent manner. Ascochlorin inhibits the mRNA expression and the protein secretion of interleukin (IL)-1β and IL-6 but not tumor necrosis factor (TNF)-α in LPS-stimulated RAW 264.7 macrophage cells. Ascochlorin suppresses nuclear translocation and DNA binding affinity of nuclear factor-κB (NF-κB). Ascochlorin down-regulates phospho-extracellular signal-regulated kinase 1/2 (p-ERK1/2) and p-p38[2].

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

Cell Viability Assay[3]

Cell Line:	HepG2, HCCLM3, Huh7 cells
Concentration:	10, 25, 50 μM
Incubation Time:	24, 48, 72 hours
Result:	Inhibit the viability of three different HCC cell lines tested (HepG2, HCCLM3 and Huh7) in a time and dose dependent manner.

Western Blot Analysis[3]

Cell Line:	HepG2 cells
Concentration:	50 μM
Incubation Time:	48 hours
Result:	Inhibited expression of the cell cycle regulator protein cyclin D1, the anti-apoptotic

	proteins Bcl-2, Mcl-1, survivin and XIAP, and the invasive gene product MMP-9.

In Vivo

Ascochlorin (Ilicicolin D) (2.5-5 mg/kg; i.p.; day 0, 1, 2, 3, 13, 15, 17, 20, 22, 24, 27, 29 and 31) inhibits tumor growth in an orthotopic HCC mouse model[1].

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

Animal Model:	Eight week-old athymic balb/c nude female mice (HCCLM3-Luc2 tumors)[3]
Dosage:	2.5 mg/kg, 5 mg/kg
Administration:	I.p.; day 0, 1, 2, 3, 13, 15, 17, 20, 22, 24, 27, 29 and 31
Result:	Induced significant inhibition of tumor growth.

REFERENCES

[1]. Min-Wen JC, et al. Molecular Targets of Ascochlorin and Its Derivatives for Cancer Therapy. Adv Protein Chem Struct Biol. 2017;108:199-225.

[2]. Lee SH, et al. Anti-Inflammatory Effect of Ascochlorin in LPS-Stimulated RAW 264.7 Macrophage Cells Is Accompanied With the Down-Regulation of iNOS, COX-2 and Proinflammatory Cytokines Through NF-κB, ERK1/2, and p38 Signaling Pathway. J Cell Biochem. 2016 Apr;117(4):978-87.

[3]. Dai X, et al. Ascochlorin, an isoprenoid antibiotic inhibits growth and invasion of hepatocellular carcinoma by targeting STAT3 signaling cascade through the induction of PIAS3. Mol Oncol. 2015 Apr;9(4):818-33.

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