ABT-510 acetate

Cat. No.: HY-13545B

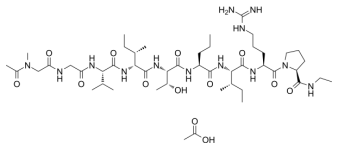
CAS No.: 442526-87-6

Molecular Formula: $C_{48}H_{87}N_{13}O_{13}$

Molecular Weight: 1054.28

Target: Apoptosis

Pathway: Apoptosis



Storage: Sealed storage, away from moisture and light, under nitrogen

Powder -80°C 2 years

-20°C 1 year

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light, under nitrogen)

SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (94.85 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	0.9485 mL	4.7426 mL	9.4851 mL
5 mM	0.1897 mL	0.9485 mL	1.8970 mL
10 mM	0.0949 mL	0.4743 mL	0.9485 mL

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

BIOLOGICAL ACTIVITY

Description

ABT-510 acetate is an anti-angiogenic TSP peptide (Thrombospondin-1 analogue) that induces apoptosis and inhibits ovarian tumour growth in an orthotopic, syngeneic model of epithelial ovarian cancer. ABT-510 acetate also reduces angiogenesis and inflammatory responses in a murine model of inflammatory bowel disease. ABT-510 acetate can be used in studies of cancer (particularly epithelial ovarian cancer) and inflammatory bowel disease (IBD)[1][2].

In Vitro

ABT-510 acetate (1, 5, 10, 20, 50 nM; 24 h) induces apoptosis in ID8 cells and increases the incidence of apoptosis in the human epithelial cancer cell lines SKOV3, OVCAR3, and CAOV3[1].

ABT-510 acetate (0-10 μM; 7 days) inhibits NO-stimulated vascular cell outgrowth into and invasion through extracellular matrix. ABT-510 acetate blocks tumor-driven vascular cell outgrowth, NO-driven cGMP flux, and CD36-mediated fatty acid uptake. [3].

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

Apoptosis Analysis[1]

Cell Line:	ID8, SKOV3, OVCAR3, and CAOV3 cells
Concentration:	1, 5, 10, 20, 50 nM
Incubation Time:	24 h
Result:	Induced ID8 cells apoptosis and increased in apoptosis in the human EOC cell lines SKOV3, OVCAR3, and CAOV3.

Cell Proliferation Assay[3]

Cell Line:	Tissue biopsies of B16F10 melanoma tumors grown in C57BL/6 mice
Concentration:	0-10 μM
Incubation Time:	7 days
Result:	Inhibited NO-stimulated vascular cell outgrowth into and invasion through extracellular matrix.

In Vivo

ABT-510 acetate (100 mg/kg; i.p.; single daily for 90 days) induces cells apoptosis in vivo and leads to a significant reduction in epithelial ovarian tumor size, ascites fluid volume, and secondary lesion dissemination in mice[1].
ABT-510 acetate (60 mg/kg; osmotic minipumps for s.c.; single daily for 7 days) decreases angiogenesis and inflammation in a murine model of inflammatory bowel disease[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	TSP-1-Null mice (C57BL/6 background; orthotopic, syngeneic model of epithelial ovarian cancer)[1]
Dosage:	100 mg/kg
Administration:	Intraperitoneal injection; single daily for 90 days
Result:	Reduced ovarian tumor growth in wild-type and TSP-1-Null Mice. Significantly reduced the volume of ascites and completely abolished the formation of peritoneal lesions. Reversed ovarian tumor hypervascularization and increased the proportion of mature blood vessels.

Animal Model:	TSP-1-Null mice (C57BL/6 background; 6-week-old; DSS-induced inflammatory bowel disease murine model)[2]
Dosage:	60 mg/kg
Administration:	Subcutaneously implanted osmotic minipumps (0.5μL/h); single daily for 7 days
Result:	Significantly delayed DSS-induced bleeding and improved the overall severity of disease. Significantly diminished inflammation grading and angiogenesis.

REFERENCES

[1]. Greenaway J, et.al. ABT-510 induces tumor cell apoptosis and inhibits ovarian tumor growth in an orthotopic, syngeneic model of epithelial ovarian cancer. Mol Cancer Ther. 2009 Jan;8(1):64-74.

[2]. Punekar S,et.al. Thrombospondin 1 and its mimetic peptide ABT-510 decrease angiogenesis and inflammation in a murine model of inflammatory bowel disease. Pathobiology. 2008;75(1):9-21.

[3]. Isenberg JS, et.al. Differential effects of ABT-510 and a CD36-binding peptide derived from the type 1 repeats of thrombospondin-1 on fatty acid uptake, nitric oxide signaling, and caspase activation in vascular cells. Biochem Pharmacol. 2008 Feb 15;75(4):875-82.

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