Coibamide A

Cat. No.:	HY-P3990
CAS No.:	1029227-48-2
Molecular Formula:	$C_{65}H_{110}N_{10}O_{16}$
Molecular Weight:	1287.63
Target:	VEGFR; Autophagy; Apoptosis
Pathway:	Protein Tyrosine Kinase/RTK; Autophagy; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.

BIOLOGICAL ACTIVITY

Description

Coibamide A, an N-methyl-stabilized cytotoxic depsipeptide, shows potent antiproliferative activity. Coibamide A induces autophagosome accumulation via an mTOR-independent mechanism. Coibamide A induces apoptosis. Coibamide A inhibits VEGFA/VEGFR2 expression and suppresses tumor growth in glioblastoma xenografts[1][2].

IC_{50} & Target

VEGFR2

In Vitro

Coibamide A (0.3-1 nM; 3-60 hours) inhibits proliferation of MDA-MB-231 breast cancer cells[1].
Coibamide A (2.3-230 nM; 3 days) produces concentration- and time-dependent cell death in human U87-MG and SF-295 glioblastoma cells[2].
Coibamide A (10-300 nM; 72 h) induces activation of caspase-3/7 and apoptosis in a cell type-specific manner[2].
Coibamide A (20 nM; 48 h) induces autophagosome accumulation in apoptotic-resistant U87-MG cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	MDA-MB-231 breast cancer cells
Concentration:	0.3, 1 nM
Incubation Time:	3-60 hours
Result:	Showed a steady concentration-dependent decrease in proliferative activity relative to vehicle-treated cells

Cell Cytotoxicity Assay[2]

Cell Line:	U87-MG and SF-295 cells
Concentration:	2.3 to 230 nM
Incubation Time:	3 days
Result:	Induced concentration-dependent cytotoxicity with EC_{50} values of 28.8 nM and 96.2 nM for U87-MG and SF-295 cells, respectively.

Apoptosis Analysis[2]

Cell Line:	U87-MG and SF-295 cells
Concentration:	10-300 nM
Incubation Time:	72 h
Result:	An 89 kDa band corresponding to the caspase 3-cleaved form of PARP1 was readily detected by 48 h indicative of apoptotic cell death in SF-295 cells, whereas only trace levels of this fragment were observed in late, detaching U87-MG cell lysates

Cell Autophagy Assay[2]

Cell Line:	U87-MG cell
Concentration:	20 nM
Incubation Time:	48 h
Result:	Caused a clear increase in LC3-II expression by 1 h, and this increase in LC3-II expression was generally sustained through 48 h.

In Vivo

Coibamide A (300 μg/kg; intratumoral injections; for the first two days, and then every 48 h afterward for 35 days) inhibits tumor growth in a subcutaneous mouse model of glioblastoma[1].

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

Animal Model:	8-week old female nude athymic mice with U87-MG cells[1]
Dosage:	300 μg/kg
Administration:	Intratumoral injections; for the first two days, and then every 48 h afterward for 35 days
Result:	Remained stable at 200-300 mm^3 without significant growth over 4 weeks of treatmen, whereas the tumors of vehicle-treated animals continued to grow at a steady rate consistent with this aggressive cancer cell type

REFERENCES

[1]. Andrew M Hau, et al. Coibamide A induces mTOR-independent autophagy and cell death in human glioblastoma cells. PLoS One. 2013 Jun 6;8(6):e65250.

[2]. Jeffrey D Serrill, et al. Coibamide A, a natural lariat depsipeptide, inhibits VEGFA/VEGFR2 expression and suppresses tumor growth in glioblastoma xenografts. Invest New Drugs. 2016 Feb;34(1):24-40.