Methylstat

Cat. No.: HY-15221

CAS No.: 1310877-95-2

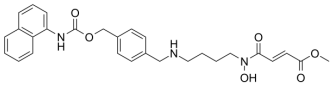
Molecular Formula: $C_{28}H_{31}N_3O_6$

Molecular Weight: 505.56

Target: Apoptosis; Histone Demethylase; MDM-2/p53

Pathway: Apoptosis; Epigenetics

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 (197.80 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.9780 mL	9.8900 mL	19.7800 mL
	5 mM	0.3956 mL	1.9780 mL	3.9560 mL
	10 mM	0.1978 mL	0.9890 mL	1.9780 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 (4.95 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Methylstat is a potent histone demethylases inhibitor. Methylstat shows anti-proliferative activity with low cytotoxicity. Methylstat induces apoptosis and cell cycle arrest at G0/G1 phase. Methylstat increases the expression of p53 and p21 protein levels. Methylstat inhibits angiogenesis induced by various cytokines. Methylstat can be used as a chemical probe for addressing its role in angiogenesis[1][2].

In Vitro

Methylstat (0-5 μM; 48, 72 h) shows anti-proliferative activity with no cytotoxicity on HUVECs at 1-2 μM[1].
Methylstat (0, 1, 2 μM; 48 h) induces cell cycle arrest at G0/G1 phase in a dose-dependent manner[1].
Methylstat (0, 1, 2 μM; 48 h) increases the expression of p53 mRNA levels, the H3K27 methylation levels and the accumulation of p53 and p21 protein levels, but suppresses the protein level of cyclinD1[1].
Methylstat (0, 1, 2 μM) shows anti-angiogenic activity induced by VEGF, bFGF and TNF-α in HUVEC cells, and inhibits the f capillary formation during CAM (chick embryo chorioallantoic membrane) development without any sign of thrombosis and hemorrhage[1].
Methylstat (1.1, 2.2 mM for U266 cells, 2.1, 4.2 mM for ARH77 cells; 72 h) induces apoptosis significantly in U266 and ARH77 cells[2].

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

Cell Cytotoxicity Assay[1]

Cell Line:	HUVEC cells
Concentration:	0-5 μM
Incubation Time:	48, 72 h
Result:	Did not exhibit cytotoxicity on HUVECs at 1-2 μM.

Cell Viability Assay[1]

Cell Line:	HUVEC, HepG2, HeLa, CHANG cells
Concentration:	0-5 μM
Incubation Time:	72 h
Result:	Showed anti-proliferative activity with IC_{50}s of 4, 10, 5, 7.5 μM for HUVEC, HepG2, HeLa, CHANG cells, respectively.

Cell Cycle Analysis[1]

Cell Line:	HUVEC cells
Concentration:	0, 1, 2 μM
Incubation Time:	48 h
Result:	G0/G1 phase increased 16.8% compared to non-treated cells, whereas S andG2/M decreased 5.5% and 6.1% respectively.

Western Blot Analysis[1]

Cell Line:	HUVEC cells
Concentration:	0, 1, 2 μM
Incubation Time:	0-48 h
Result:	Resulted in accumulation of p53 and p21 protein levels in a time- and dose-dependent manner and increased the H3K27 methylation levels, the but suppressed the protein level of cyclinD1.

Apoptosis Analysis[2]

Cell Line:	U266, ARH77 cells
Concentration:	1.1, 2.2 mM for U266 cells, 2.1, 4.2 mM for ARH77 cells
Incubation Time:	72 h
Result:	Induced apoptosis in U266, ARH77 cells.

REFERENCES

[1]. Yumi Cho, et al. A histone demethylase inhibitor, methylstat, inhibits angiogenesis in vitro and in vivo. RSC Advances, 2014.

[2]. Kacı FN, et al. Synergistic Apoptotic Effects of Bortezomib and Methylstat on Multiple Myeloma Cells. Arch Med Res. 2020 Apr;51(3):187-193.

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