Cerivastatin sodium

Cat. No.: HY-109523

CAS No.: 143201-11-0

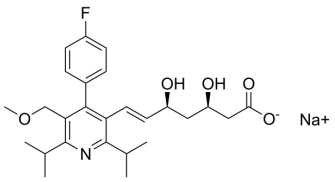
Molecular Formula: $C_{26}H_{33}FNNaO_5$

Molecular Weight: 481.53

Target: HMG-CoA Reductase (HMGCR); Ferroptosis

Pathway: Metabolic Enzyme/Protease; Apoptosis

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



SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (207.67 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0767 mL	10.3836 mL	20.7671 mL
	5 mM	0.4153 mL	2.0767 mL	4.1534 mL
	10 mM	0.2077 mL	1.0384 mL	2.0767 mL

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

BIOLOGICAL ACTIVITY

Description

Cerivastatin sodium is a synthetic lipid-lowering agent and a highly potent, well-tolerated and orally active HMG-CoA reductase inhibitor, with a Ki of 1.3 nM/L. Cerivastatin sodium reduces low-density lipoprotein cholesterol levels. Cerivastatin sodium also inhibits proliferation and invasiveness of MDA-MB-231 cells, mainly by RhoA inhibition, and has anti-cancer effect[1][2].

IC_{50} & Target

Ki: 1.3 nM/L (HMG-CoA reductase)[1][2][3]

In Vitro

Cerivastatin sodium (5-50 ng/mL; 3 days; MDA-MB-231 cells) treatment induces a dose-dependent decrease in cell proliferation of MDA-MB-231 cells (up to 40% inhibition at 25 ng/mL)[1].

Cerivastatin sodium (25 ng/mL; 18-36 hours; MDA-MB-231 cells) treatment induces an arrest of the cell cycle in G 1/S phase after 36 h treatment. This arrest is not observed for a shorter incubation time (18 h)[1].

Cerivastatin sodium (25 ng/mL; 18 hours; MDA-MB-231 cells) treatment induces a marked increase in the level of p21 $^{Waf1/Cip1}$[1].

Cerivastatin sodium (25 ng/mL; 12 hours; MDA-MB-231 cells) treatment increases the p21 transcript in MDA-MB-231 cells[1].

Cerivastatin sodium (10-25 ng/mL; 18 hours) inhibits invasion of MDA-MB-231 cells through Matrigel[1].

Cerivastatin sodium (25 ng/mL; 18-36 hours) delocalizes RhoA and Ras from the membrane to the cytosol and induces

morphological changes[1].
Cerivastatin sodium (25 ng/mL; 4-36 hours) induces inactivation of NFκB, in a RhoA inhibition-dependent manner, resulting in a decrease in urokinase and metalloproteinase-9 expression, and concomitantly increases IκB[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	MDA-MB-231 cells
Concentration:	5 ng/mL, 10 ng/mL, 25 ng/mL, 50 ng/mL
Incubation Time:	3 days
Result:	Induced a dose-dependent decrease in cell proliferation of MDA-MB-231 cells.

Cell Cycle Analysis[1]

Cell Line:	MDA-MB-231 cells
Concentration:	25 ng/mL
Incubation Time:	18 hours, 36 hours
Result:	Induced a cell cycle block in G 1/S phase.

Western Blot Analysis[1]

Cell Line:	MDA-MB-231 cells
Concentration:	25 ng/mL
Incubation Time:	18 hours
Result:	Induced a marked increase in the level of $p21^{Waf1/Cip1}$.

RT-PCR[1]

Cell Line:	MDA-MB-231 cells
Concentration:	25 ng/mL
Incubation Time:	12 hours
Result:	Increased $p21^{Waf1/Cip1}$ mRNA levels.

In Vivo

Cerivastatin sodium is well absorbed, reaching maximal plasma levels in 1-3 hours following oral dosing. In the circulation, Cerivastatin sodium is highly bound to plasma proteins (99.5%), with an elimination half-life of 2-4 hours. Cerivastatin is metabolized predominantly in the liver to three polar metabolites. Two of these metabolites are active, but to a lesser extent compared to parent drug, and the third metabolite is inactive. Plasma concentrations of all metabolites are substantially lower than those of the parent drug. Elimination of metabolites is via the urine (20-25%) and feces (66-73%), while essentially no parent compound is excreted[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Denoyelle C, et al. Cerivastatin, an inhibitor of HMG-CoA reductase, inhibits the signaling pathways involved in the invasiveness and metastatic properties of highly invasive breast cancer cell lines: an in vitro study. Carcinogenesis. 2001 Aug;22(8):1139-48.

[2]. Stein E, et al. Cerivastatin, a New Potent Synthetic HMG Co-A Reductase Inhibitor: Effect of 0.2 mg Daily in Subjects With Primary Hypercholesterolemia. J Cardiovasc Pharmacol Ther. 1997 Jan;2(1):7-16.

[3]. Furberg CD, et al. Withdrawal of cerivastatin from the world market. Curr Control Trials Cardiovasc Med. 2001;2(5):205-207.

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