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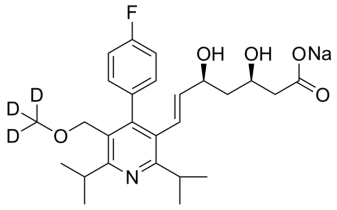
Cerivastatin-d_3 sodium

Cat. No.:	HY-109523S
CAS No.:	916314-45-9
Molecular Formula:	$C_{26}H_{30}D_3FNNaO_5$
Molecular Weight:	484.55
Target:	Ferroptosis; HMG-CoA Reductase (HMGCR); Isotope-Labeled Compounds
Pathway:	Apoptosis; Metabolic Enzyme/Protease; Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Cerivastatin-d_3 sodium is deuterated labeled Cerivastatin sodium (HY-109523). 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].

In Vitro

Stable heavy isotopes of hydrogen, carbon, and other elements have been incorporated into drug molecules, largely as tracers for quantitation during the drug development process. Deuteration has gained attention because of its potential to affect the pharmacokinetic and metabolic profiles of drugs[1].

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)[2].

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)[2].

Cerivastatin sodium (25 ng/mL; 18 hours; MDA-MB-231 cells) treatment induces a marked increase in the level of p21 $^{Waf1/Cip1}$[2].

Cerivastatin sodium (25 ng/mL; 12 hours; MDA-MB-231 cells) treatment increases the p21 transcript in MDA-MB-231 cells[2].

Cerivastatin sodium (10-25 ng/mL; 18 hours) inhibits invasion of MDA-MB-231 cells through Matrigel[2].

Cerivastatin sodium (25 ng/mL; 18-36 hours) delocalizes RhoA and Ras from the membrane to the cytosol and induces morphological changes[2].

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[2].

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

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[3].

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.

[4]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. Ann Pharmacother. 2019 Feb;53(2):211-216.

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