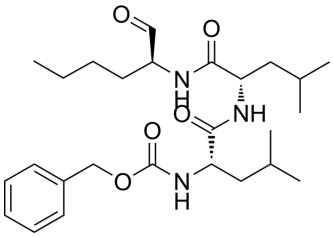
Z-LLNle-CHO

Cat. No.:	HY-120234
CAS No.:	133407-83-7
Molecular Formula:	$C_{26}H_{41}N_3O_5$
Molecular Weight:	475.62
Target:	γ-secretase; Proteasome; Apoptosis
Pathway:	Neuronal Signaling; Stem Cell/Wnt; Metabolic Enzyme/Protease; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Z-LLNle-CHO (Z-Leu-Leu-Nle-CHO) is a γ-secretase inhibitor I. Z-LLNle-CHO induces caspase and ROS-dependent apoptosis by blocking the Akt-mediated pro-survival pathway. Z-LLNle-CHO can be used in cancer research, such as breast cancer and leukaemia[1][2].

In Vitro

Z-LLNle-CHO (0-5 μM or 0-3 μM; 72 h) results in a dose-dependent decrease in cell viability/proliferation in six breast cancer cell lines[1].

Z-LLNle-CHO shows proteasome inhibitory activity, which contributes to cytotoxicity to MCF-7 cells[1].

Z-LLNle-CHO blocks Akt-mediated pro-survival pathways and induces caspase- and ROS-dependent cell apoptosis in Nalm6 and 697 cells[2].

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

Cell Viability Assay[1]

Cell Line:	MCF-7, BT474, T47D, MDA-MB-231, SKBR3, and MDA-MB-468 cells
Concentration:	0-5 μM (for MCF-7); 0-3 μM
Incubation Time:	72 h
Result:	Inhibited MCF-7, BT474, T47D, MDA-MB-231, SKBR3, and MDA-MB-468 cells with ED_{50} values of 3.25, 2.5, 2.4, 1.8, 1.6, and 1.4 μM, respectively.

Apoptosis Analysis[2]

Cell Line:	Precursor-B ALL cells
Concentration:	0-2.5 μM
Incubation Time:	18-24 h
Result:	Induced cell apoptosis.

In Vivo

Z-LLNle-CHO (5 mg/kg; s.c.; single daily for 12 days) inhibits engraftment of B-lymphoblasts in precursor-B ALL xenograft model[2].

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Animal Model:	Female SCID/NOD mice (6-8-week-old; precursor-B ALL xenograft model)[2].
Dosage:	5 mg/kg
Administration:	Subcutaneous injection; single daily for 12 days
Result:	Delayed or prevented engraftment of B-lymphoblasts in 50% of the animals comprising the experimental group.

REFERENCES

[1]. Han J, et al. The cytotoxicity of gamma-secretase inhibitor I to breast cancer cells is mediated by proteasome inhibition, not by gamma-secretase inhibition. Breast Cancer Res. 2009;11(4):R57.

[2]. Meng X, et al. GSI-I (Z-LLNle-CHO) inhibits γ-secretase and the proteosome to trigger cell death in precursor-B acute lymphoblastic leukemia. Leukemia. 2011 Jul;25(7):1135-46.

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