L67

Cat. No.: HY-15586

CAS No.: 325970-71-6

Molecular Formula: $C_{16}H_{14}Br_2N_4O_4$

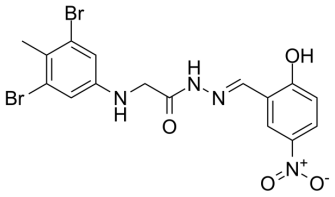
Molecular Weight: 486.11

Target: DNA/RNA Synthesis; Caspase; Apoptosis; Reactive Oxygen Species

Pathway: Cell Cycle/DNA Damage; Apoptosis; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (205.71 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	2.0571 mL	10.2857 mL	20.5715 mL
5 mM	0.4114 mL	2.0571 mL	4.1143 mL
10 mM	0.2057 mL	1.0286 mL	2.0571 mL

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

BIOLOGICAL ACTIVITY

Description

L67 (DNA Ligase Inhibitor) is a competitive DNA ligase inhibitor that effectively inhibits DNA ligases I/III (both IC_{50} are 10 μM). L67 (DNA Ligase Inhibitor) can cause nuclear DNA damage by reducing levels of mitochondrial DNA and increasing levels of mitochondrially-generated ROS. L67 (DNA Ligase Inhibitor) also activates the Caspase 1-dependent apoptosis pathway in cancer cells, can be used in cancer research[1][2].

In Vitro

L67 (10, 15 μM; 24 h) promotes nuclear DNA damage and (0-50 μM) increases level of mSOX by inhibiting mitochondrial LigIII α in HeLa cells[1].

L67 (10 μM; 24 h) induces changes in mitochondrial function that causes a reduction in OCR and mitochondrial DNA, and abnormal mitochondrial morphology in HeLa[1].

L67 (10, 100 μM; 24 h) induces apoptosis in cancer cells[1].

L67 (0-30 μM; 24 h) selectively induces cell death in cancer cells by activating a caspase 1-dependent apoptotic pathway[1].

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

Cell Viability Assay[1]

Cell Line:	HeLa cells
Concentration:	10, 15 μM; 0-50 μM
Incubation Time:	24 h
Result:	Increased the formation of nuclear γH2AX foci and steady state levels of γH2AX when at 10 or 15 μM. (γH2AX: a sign of DNA double-strand breaks). Resulted in a concentration (0-50 μM)-dependent increase in mSOX (mitochondrial superoxide) levels. (mSOX is a major cause of the cellular oxidative damage).

Cell Viability Assay[1]

Cell Line:	HeLa cells
Concentration:	10 μM
Incubation Time:	24 h
Result:	Reduced oxygen consumption rate (OCR) approximately 20%. Resulted in about a 25% reduction in mitochondrial DNA.

Apoptosis Analysis[1]

Cell Line:	HeLa cells
Concentration:	10, 100 μM
Incubation Time:	24 h
Result:	Result:Induced apoptosis, and at 100 μM with apoptotic cells constituting about 50% of the HeLa cell population.

Cell Viability Assay[1]

Cell Line:	HeLa cells
Concentration:	0-30 μM
Incubation Time:	24 h
Result:	Activates a caspase 1-dependent cell death pathway in cancer cells.

CUSTOMER VALIDATION

- Nat Methods. 2023 Jul 20.

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REFERENCES

[1]. Sallmyr A, et al. Inhibiting Mitochondrial DNA Ligase IIIα Activates Caspase 1-Dependent Apoptosis in Cancer Cells. Cancer Res. 2016 Sep 15;76(18):5431-41.

[2]. Chen X, et al. Rational design of human DNA ligase inhibitors that target cellular DNA replication and repair. Cancer Res. 2008 May 1;68(9):3169-77.

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