PRDX1-IN-1

Cat. No.: HY-149394

Molecular Formula: $C_{46}H_{55}N_3O_4$

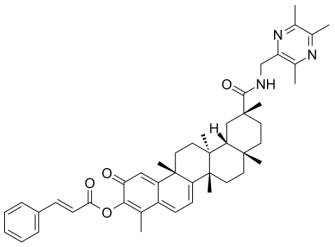
Molecular Weight: 713.95

Target: Apoptosis; ROS Kinase

Pathway: Apoptosis; Protein Tyrosine Kinase/RTK

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (140.07 mM; Need ultrasonic)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	1.4007 mL	7.0033 mL	14.0066 mL
5 mM	0.2801 mL	1.4007 mL	2.8013 mL
10 mM	0.1401 mL	0.7003 mL	1.4007 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 (3.50 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.5 mg/mL (3.50 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

PRDX1-IN-1 is a selective inhibtor of PRDX1 with an IC_{50} value of 0.164 μM. PRDX1-IN-1 can be used in researches related to cancer.PRDX1-IN-1 promotes intracellular ROS accumulation, and inhibits the proliferation, invasion and migration of cancer cells besides inducing apoptosis. PRDX1-IN-1 could be used in cancer research[1].

IC_{50} & Target

IC50:0.164μM(potent peroxiredoxin 1,PRDX1)[1]

In Vitro

PRDX1-IN-1 inhibites the proliferation activities of the human lung cancer cells A549, lung cancer cell lines (LTEP-a-2 and H1975), human breast cancer cell line (MDA-MB-231), human hepatoma cell line (SK-Hep-1) with the IC_{50} values of 1.92 μM, 2.93 μM, 1.99 μM, 2.67 μM, 2.42μM, respectively[1].

PRDX1-IN-1 (compound 7e)(2 μM or 4 μM, 24 h) promotes intracellular ROS accumulation, and inhibits the invasion and migration of human lung cancer cells A549[1].

PRDX1-IN-1 (2 μM or 4 μM, 24 h) induces the apoptosis of A549 cells[1].
PRDX1-IN-1 (2 μM or 4 μM, 6 h) suppresses the key signaling pathways (AKT and ERK) and promotes the expression of apoptosis-related proteins (cleaved caspase-3/8 and cleaved PARP) in A549 cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Proliferation assay[1]

Cell Line:	lung cancer cell lines (LTEP-a-2 and H1975), human breast cancer cell line (MDA-MB-231), human hepatoma cell line (SK-Hep-1)
Concentration:	0.5–10 μM
Incubation Time:	48 h
Result:	Inhibited the proliferation activities of cancer cells A549, LTEP-a-2, H1975, MDA-MB-231, SK-Hep-1.

Apoptosis assay [1]

Cell Line:	human lung cancer cells A549
Concentration:	2 μM or 4 μM
Incubation Time:	24 h
Result:	Increased the ratio of the total number of early (annexin-V+/PI−) and late (annexin-V+/PI+) apoptotic cells significantly.

WB assay[1]

Cell Line:	A549 cell
Concentration:	2 μM or 4 μM
Incubation Time:	6 h
Result:	Decreased the phosphorylation levels of PI3K, AKT, C-RAF and ERK.

Matrigel invasion assay[1]

Cell Line:	A549 cell
Concentration:	2 μM or 4 μM
Incubation Time:	24 - 48 h
Result:	Inhibted the cell matrigel and invasion.

In Vivo

PRDX1-IN-1 (0.5 or 1 mg/kg, intraperitoneal(i.p.), 19 days, every day) inhibited tumor growth in a mouse model of lung cancer[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57BL/6J male mice injected with Lewis cell (lung cancer)[1]
Dosage:	0.5 or 1 mg/kg
Administration:	intraperitoneal injection (i.p.), every day for 19 days.
Result:	Inhibited tumor growth, with the tumor growth inhibition (TGI) values of 77.47% and

	69.89% in the groups of 1 mg/kg and 0.5 mg/kg, respectively. Induced the changes in morphological characteristics of tumor cells, including cell agglutination, contraction, and nuclear chromatin marginalization.

REFERENCES

[1]. Ying Bai, et al. Development of novel celastrol-ligustrazine hybrids as potent peroxiredoxin 1 inhibitors against lung cancer. Eur J Med Chem. 2023, 259, 115656.

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