Didesmethylrocaglamide

Cat. No.: HY-19356A

CAS No.: 177262-30-5

Molecular Formula: $C_{27}H_{27}NO_7$

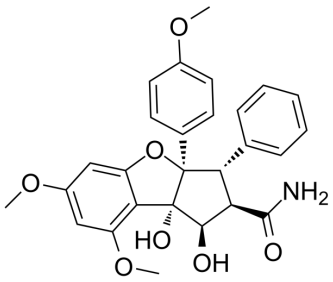
Molecular Weight: 477.51

Target: Eukaryotic Initiation Factor (eIF); Apoptosis

Pathway: Cell Cycle/DNA Damage; Apoptosis

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 (209.42 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0942 mL	10.4710 mL	20.9420 mL
	5 mM	0.4188 mL	2.0942 mL	4.1884 mL
	10 mM	0.2094 mL	1.0471 mL	2.0942 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 (5.24 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.24 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.24 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Didesmethylrocaglamide, a derivative of Rocaglamide, is a potent eukaryotic initiation factor 4A (eIF4A) inhibitor. Didesmethylrocaglamide has potent growth-inhibitory activity with an IC_{50} of 5 nM. Didesmethylrocaglamide suppresses multiple growth-promoting signaling pathways and induces apoptosis in tumor cells. Antitumor activity[1][2].

IC_{50} & Target

Eukaryotic initiation factor 4A (eIF4A)[1]

In Vitro

Didesmethylrocaglamide (5 nM, and 10 nM; 72 hours; MPNST cells) treatment arrests MPNST cells at G2-M, increases the sub-

G1 population, induces cleavage of caspases and PARP, and elevates the levels of the DNA-damage response marker γH2A.X, while decreasing the expression of AKT and ERK1/2[1].
Didesmethylrocaglamide inhibits MPNST cell proliferation by inducing cell cycle arrest at G2/M and subsequently, cell death.
Didesmethylrocaglamide-treated 697-R cells exhibits IC_{50} values is very similar to those of parental 697 cells (4 vs 3nM of IC_{50}, respectively)[1].
Didesmethylrocaglamide induces apoptosis in both neurofibromatosis type 1 (NF1)-expressing and NF1-deficient MPNST cells, possibly subsequent to the activation of the DNA damage response. Didesmethylrocaglamide-treated sarcoma cells show decreased levels of multiple oncogenic kinases, including insulin-like growth factor-1 receptor[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	Malignant peripheral nerve sheath tumors (MPNST) cells
Concentration:	5 nM, and 10 nM
Incubation Time:	72 hours
Result:	Induced cleavage of caspases and PARP, and elevated the levels of the DNA-damage response marker γH2A.X.

REFERENCES

[1]. Long-Sheng Chang, et al. Targeting Protein Translation by Rocaglamide and Didesmethylrocaglamide to Treat MPNST and Other Sarcomas. Mol Cancer Ther. 2020 Mar;19(3):731-741.

[2]. Long-Sheng Chang, et al. Abstract 1950: The eIF4A inhibitors didesmethylrocaglamide and rocaglamide as effective treatments for pediatric bone and soft-tissue sarcomas. Cancer Res 2020;80(16 Suppl):Abstract nr 1950.

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