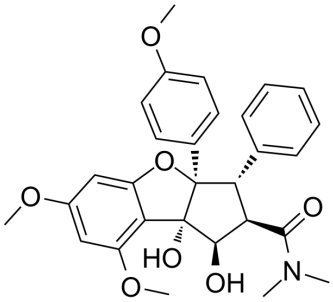
Rocaglamide

Cat. No.:	HY-19356
CAS No.:	84573-16-0
Molecular Formula:	$C_{29}H_{31}NO_7$
Molecular Weight:	505.56
Target:	Eukaryotic Initiation Factor (eIF); HSP; NF-κB
Pathway:	Cell Cycle/DNA Damage; 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 (197.80 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.9780 mL	9.8900 mL	19.7800 mL
	5 mM	0.3956 mL	1.9780 mL	3.9560 mL
	10 mM	0.1978 mL	0.9890 mL	1.9780 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: ≥ 7.5 mg/mL (14.84 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 7.5 mg/mL (14.84 mM); Clear solution
3. Add each solvent one by one: 5% DMSO >> 95% saline
   Solubility: ≥ 4.76 mg/mL (9.42 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Rocaglamide (Roc-A) is isolated from the genus Aglaia and can be used for coughs, injuries, asthma and inflammatory skin diseases. Rocaglamide is a potent inhibitor of NF-κB activation in T-cells. Rocaglamide is a potent and selective heat shock factor 1 (HSF1) activation inhibitor with an IC_{50} of ~50 nM. Rocaglamide inhibits the function of the translation initiation factor eIF4A. Rocaglamide also has anticancer properties in leukemia[1][2][3].

IC_{50} & Target		
	eIF4	HSF1 50 nM (IC_{50})

In Vitro	Rocaglamide enhances TRAIL-induced apoptosis in resistant HCC cells. Treatment with Rocaglamide alone leads to apoptosis in 9% HepG2 and 11% Huh-7 cells and treatment with TRAIL induces apoptosis in 16% HepG2 and 17% Huh-7 cells. However, the combination of Rocaglamide and TRAIL induces apoptosis in 55% HepG2 and 57% Huh-7 cells, which is evidently more than an additive effect. A similar result is obtained by measurement of cell viability using crystal violet staining. Rocaglamide has the potential to sensitize highly chemoresistant HepG2 and Huh-7 cells to TRAIL-based therapy[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Tumor volumes in the Rocaglamide-treated group are 45±12% compared with the control group. Rocaglamide significantly suppresses tumor growth compared with that in the control group. Treatment with Rocaglamide does not lead to any reduction in body weight and no apparent signs of toxicity are observed in the mice during the treatment, suggesting that Rocaglamide is generally tolerated well[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	HepG2 and Huh-7 cells (1×10^4/well) are seeded in 96-well plates in complete culture medium and incubated for 24 h. The cells are then exposed to 100 nM Rocaglamide and/or 100 ng/mL TRAIL for 24 h. The control cells are treated with DMSO at a concentration equal to that used for the drug-treated cells. The complete culture medium is then removed and MTT (200 μL, 0.5 mg/mL in 10% FBS-containing DMEM) is added to each well and the plate is incubated for 2 h at 37°C in a humidified incubator. The solution is then removed from the wells and 200 μL DMSO is added to each well prior to agitation. The absorbance at 570 nm is read using a microplate reader (Bio-Tek ELx800). The value for the vehicle-treated cells is considered to indicate 100% viability. Furthermore, a crystal violet assay is carried out. Briefly, the cells (1×10^5/mL) are seeded in a 12 well plate for 12 h, and treated with TRAIL (0-100 ng/mL) and/or RocA(1-100 nM) for 12 h. The treated cells are washed with phosphate-buffered saline (PBS), fixed with 4% paraformaldehyde for 15 min, and stained using crystal violet for a further 30 min[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] The Huh-7 cells (3×10^6), suspended in 100 μL mix (equal volumes of DMEM and Matrigel), are implanted subcutaneously into the right flank of 10 female SCID mice (6-week-old) and then randomly divided into two equal groups, one of which received an intraperitoneal injection of Rocaglamide (2.5 mg/kg in 80 μL olive oil; n=5) and the other, used as a vehicle control, received olive oil alone (n=5). These treatments are performed once daily for 32 days and the tumor volumes and body weights of the animals are measured twice a week. The tumor volumes (mm^3) are calculated using the following formula: Tumor volume=$LS^2/2$, where L is the longest diameter and S is the shortest. At the end of the experiments, the mice are sacrificed and tumor samples are harvested, fixed in formalin and embedded in paraffin as tissue sections for immunohistochemical analysis. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Commun. 2023 Feb 2;14(1):553.
- Nat Commun. 2021 Jun 17;12(1):3720.
- Autophagy. 2020 Mar;16(3):419-434.
- Cell Rep. 2021 Oct 12;37(2):109806.
- Cell Mol Life Sci. 2021 Aug 16.

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REFERENCES

[1]. Santagata S, et al. Tight coordination of protein translation and heat shock factor 1 activation supports the anabolic malignant state. Science. 2013 Jul 19; 341(6143): 1238303.

[2]. Luan Z, et al. Rocaglamide overcomes tumor necrosis factor-related apoptosis-inducing ligand resistance in hepatocellular carcinoma cells by attenuating the inhibition of caspase-8 through cellular FLICE-like-inhibitory protein downregulation. Mol Med Rep

[3]. Baumann B, et al. Rocaglamide derivatives are potent inhibitors of NF-kappa B activation in T-cells. J Biol Chem. 2002 Nov 22;277(47):44791-800.

Caution: Product has not been fully validated for medical applications. For research use only.

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