9-cis-Retinoic acid

Cat. No.: HY-15128

CAS No.: 5300-03-8

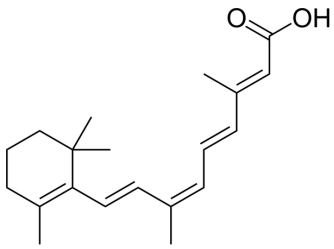
Molecular Formula: $C_{20}H_{28}O_2$

Molecular Weight: 300.44

Target: RAR/RXR; Apoptosis; Endogenous Metabolite

Pathway: Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor; Apoptosis

Storage: -20°C, protect from light, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light, stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (83.21 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.3285 mL	16.6423 mL	33.2845 mL
	5 mM	0.6657 mL	3.3285 mL	6.6569 mL
	10 mM	0.3328 mL	1.6642 mL	3.3285 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 (8.32 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (8.32 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

9-cis-Retinoic acid (ALRT1057), a vitamin A derivative, is a potent RAR/RXR agonist. 9-cis-Retinoic acid induces apoptosis, regulates cell cycle and has anticancer, anti-inflammatory and neuroprotection activities[1][2][3][4][5].

IC_{50} & Target

Human Endogenous Metabolite

In Vitro

9-cis-Retinoic acid (1-10 μM; 0-5 days; CA 9-22 and NA cells) treatment significantly decreases proliferation in a dose-dependent manner in CA 9-22 and NA cells[1].

9-cis-Retinoic acid (1 μM; 24 hours) treatment significantly increases PPARγ functional activity by >200% in CA 9-22 and NA aerodigestive cells[1].

9-cis-Retinoic acid treatment results in the formation of a nuclear PPARγ-RXRα heterodimer supershift complex in CA 9-22 cells[1].

9-cis-Retinoic acid inhibits proliferation and induces apoptosis in cutaneous T-cell lymphoma (CTCL) in a dose-dependent and time-dependent manner. 9-cis-Retinoic acid also induces G0/G1 cell cycle arrest by downregulation of cyclin D1. 9-cis-Retinoic acid significantly decreases phosphorylation of JAK1, STAT3, and STAT5 and downregulated Bcl-xL and cyclin D1[2].

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

Cell Proliferation Assay[1]

Cell Line:	CA 9-22 and NA cells
Concentration:	1 μM, 10 μM
Incubation Time:	0 day, 1 day, 3 days, 5 days
Result:	Significantly decreased proliferation.

In Vivo

9-cis-Retinoic acid (1 mg/kg; intravenous injection; daily; for 10 days; male C57BL/6J mice) treatment significantly decreases the serum ALT and AST level, alleviates hepatic necrosis of the bile duct ligation (BDL)-mice[3].

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

Animal Model:	Male C57BL/6J mice (6-8 weeks; 19-22 g) treatment with bile duct ligated[3]
Dosage:	1 mg/kg
Administration:	Intravenous injection; daily; for 10 days
Result:	Significantly decreased the serum ALT and AST level, alleviated hepatic necrosis.

REFERENCES

[1]. Raul Rosas, et al. Retinoids Augment Thiazolidinedione PPARγ Activation in Oral Cancer Cells. Anticancer Res. 2020 Jun;40(6):3071-3080.

[2]. Hua Yang, et al. Effects of 9-cis-retinoic Acid on the Proliferation and Apoptosis of Cutaneous T-cell Lymphoma Cells. Anticancer Drugs. 2019 Jan;30(1):56-64.

[3]. Zhiqing Yuan, et al. 9-cis-retinoic Acid Elevates MRP3 Expression by Inhibiting Sumoylation of RXRα to Alleviate Cholestatic Liver Injury. Biochem Biophys Res Commun. 2018 Sep 3;503(1):188-194.

[4]. V M Manzano, et al. Human Renal Mesangial Cells Are a Target for the Anti-Inflammatory Action of 9-cis Retinoic Acid. Br J Pharmacol. 2000 Dec;131(8):1673-83.

[5]. Gro H Mathisen, et al. Delayed Translocation of NGFI-B/RXR in Glutamate Stimulated Neurons Allows Late Protection by 9-cis Retinoic Acid. Biochem Biophys Res Commun. 2011 Oct 14;414(1):90-5.

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