# 9-cis-Retinoic acid

**Cat. No.:** HY-15128

**CAS No.:** 5300-03-8

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

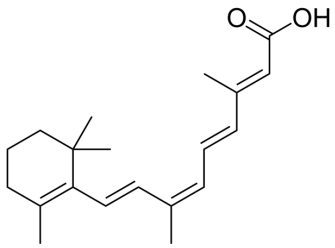
**Molecular Weight:** 300.44

**Target:** RAR/RXR; Apoptosis

**Pathway:** Metabolic Enzyme/Protease; Apoptosis

**Storage:** -20°C, stored under nitrogen

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




## SOLVENT & SOLUBILITY

**In Vitro**

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

| Preparing Stock Solutions | Solvent / Mass / 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].

**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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