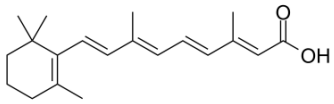
Retinoic acid

Cat. No.:	HY-14649
CAS No.:	302-79-4
Molecular Formula:	$C_{20}H_{28}O_2$
Molecular Weight:	300.44
Target:	RAR/RXR; PPAR; Endogenous Metabolite; Autophagy
Pathway:	Metabolic Enzyme/Protease; Cell Cycle/DNA Damage; Autophagy
Storage:	-20°C, sealed storage, away from moisture and light * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (166.42 mM)
H_2O : < 0.1 mg/mL (insoluble)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent \ Mass	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 and warming
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
3. Add each solvent one by one: **10% DMSO >> 90% corn oil**
   Solubility: ≥ 2.5 mg/mL (8.32 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Retinoic acid is a metabolite of vitamin A that plays important roles in cell growth, differentiation, and organogenesis. Retinoic acid is a natural agonist of **RAR** nuclear receptors, with **IC_{50}**s of 14 nM for RARα/β/γ. Retinoic acid bind to **PPARβ/δ** with **K_d** of 17 nM. Retinoic acid acts as an inhibitor of transcription factor Nrf2 through activation of retinoic acid receptor alpha.

IC_{50} & Target	PPARβ/δ 17 nM (Kd)	PPARα 103 nM (Kd)	PPARγ 178 nM (Kd)	RARα 14 nM (IC_{50})
	RARβ 14 nM (IC_{50})	RARγ 14 nM (IC_{50})	Human Endogenous Metabolite	

In Vitro

Retinoic acid (All-trans-retinoic acid, ATRA) is a highly potent derivative of vitamin A that is required for virtually all essential physiological processes and functions because of its involvement in transcriptional regulation of over 530 different genes. Retinoic acid exerts its actions by serving as an activating ligand of nuclear retinoic acid receptors (RARα-γ), which form heterodimers with retinoid X receptors (RXRα-γ)[1].

Retinoic acid (RA) bound to PPARα and PPARγ with a low affinity demonstrated by K_d values of 100-200 nM. In contrast, Retinoic acid associates with PPARβ/δ with a K_d of 17 nM, revealing both high affinity and isotype selectivity [2].

Undifferentiated P19 cells express the Retinoic acid (RA) receptors RARα, RARβ, RARγ, and PPARβ/δ, as well as the Retinoic acid -binding proteins CRABP-II and FABP5. Induction of differentiation by treatment of cells with Retinoic acid results in transient up-regulation of CRABP-II and down-regulation of FABP5 that are observed at the level of both the respective proteins and mRNAs. Following the initial decrease, the level of both FABP5 protein and mRNA increases to attain a 2-2.5-fold higher level in mature neurons as compared with undifferentiated P19 cells. Induction of differentiation does not markedly affect the levels of either RARα or PPARβ/δ. The level of RARγ mRNA decreases by about 5-fold by day 4 and remained low in mature neurons[3].

Retinoic acid (RA) is a morphogen derived from retinol (vitamin A) that plays important roles in cell growth, differentiation, and organogenesis. The Retinoic acid interacts with retinoic acid receptor (RAR) and retinoic acid X receptor (RXR) which then regulate the target gene expression[4].

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

PROTOCOL

Cell Assay [3]

P19 cell are induced to undergo neuronal differentiation according to established procedures. Briefly, cells are cultured on 1% agarose-coated 10 cm dishes at 3×10^5 cells/mL in α-minimal essential medium supplemented with 10% FBS. Differentiation is induced by addition of Retinoic acid (1 μM) and medium containing Retinoic acid replaced 2 days later. On day 4, cell aggregates are collected by centrifugation, separated to single cells by trypsin/EDTA treatment, replated onto poly-L-lysine-coated plates, and cultured in α-minimal essential medium supplemented with 10% FBS. On day 6, medium is replaced with neurobasal medium containing B27 supplement and 2 mM GlutaMAX. Medium is replaced every 2 days for an additional week[3].

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

CUSTOMER VALIDATION

- **Cancer Lett**. 2020 Mar 31;473:130-138.
- **BBA-Mol Basis Dis**. 2019 Oct.
- **PLoS Negl Trop Dis**. 2019 Aug 20;13(8):e0007681.
- **Life Sci**. 2019 Dec 30:117247.
- **Eur J Pharmacol**. 2019 May 15;851:174-185.

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REFERENCES

[1]. Wu L, et al. Retinoid X Receptor Agonists Upregulate Genes Responsible for the Biosynthesis of All-Trans-Retinoic Acid in Human Epidermis. PLoS One. 2016 Apr 14;11(4):e0153556.

[2]. Shaw N, et al. Retinoic acid is a high affinity selective ligand for the peroxisome proliferator-activated receptor beta/delta. J Biol Chem. 2003 Oct 24;278(43):41589-92.

[3]. Yu S, et al. Retinoic acid induces neurogenesis by activating both retinoic acid receptors (RARs) and peroxisomeproliferator-activated receptor β/δ (PPAR β/δ). J Biol Chem. 2012 Dec 7;287(50):42195-205.

[4]. Kam RK, et al. Retinoic acid synthesis and functions in early embryonic development. Cell Biosci. 2012 Mar 22;2(1):11.

[5]. Apfel C, et al. A retinoic acid receptor alpha antagonist selectively counteracts retinoic acid effects. Proc Natl Acad Sci U S A. 1992 Aug 1;89(15):7129-33.

[6]. Xiu Jun Wang, et al. Identification of retinoic acid as an inhibitor of transcription factor Nrf2 through activation of retinoic acid receptor alpha. Proc Natl Acad Sci U S A. 2007 Dec 4;104(49):19589-94.

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