Adapalene

Cat. No.: HY-B0091

CAS No.: 106685-40-9

Molecular Formula: $C_{28}H_{28}O_3$

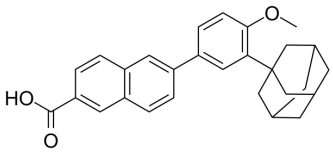
Molecular Weight: 412.52

Target: RAR/RXR; Autophagy; Apoptosis

Pathway: Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor; Autophagy; 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 : 10 mg/mL (24.24 mM; ultrasonic and warming and heat to 60°C)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	2.4241 mL	12.1206 mL	24.2412 mL
5 mM	0.4848 mL	2.4241 mL	4.8482 mL
10 mM	0.2424 mL	1.2121 mL	2.4241 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: 1 mg/mL (2.42 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1 mg/mL (2.42 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Adapalene (CD271), a third-generation synthetic retinoid, is widely used for the research of acne. Adapalene is a potent RAR agonist, with AC_{50}s of 2.3 nM, 9.3 nM, and 22 nM for RARβ, RARγ, RARα, respectively. Adapalene also inhibits the enzymatic activity of GOT1 in a non-competitive manner. Adapalene exhibits anti-tumor activity[1][2][3].

IC_{50} & Target

AC50: 2.3 nM (RARβ), 9.3 nM (RARγ), and 22 nM (RARα)[1]

In Vitro

Adapalene (1-200 μM; 24 h) inhibits the viability of ES-2, HOV-7, MCF-7 , Hela, SW1990, HT1080, and MM-468 cells, with IC_{50}s of 10.36 μM, 10.81 μM, 12.00 μM, 19.08 μM, 19.52 μM, 21.70 μM, and 31.47 μM, respectively[2].

Adapalene (10-40 μM; 24 h) induces ES-2 cells apoptosis and inhibits proliferation in vitro[2].
Adapalene (3-30 μM; 6-24 h) significantly increases the G1-phase population in LoVo or DLD1 cells[3].
Adapalene (1-200 μM) inhibits GOT1 activity, with an IC_{50} of 21.79 μM[2].
Adapalene (10^{-6}-10^{-3} nM) inhibits the expression of plasma membrane-associated enzyme transglutaminase Type I, with an IC_{50} of 2.5 nM[1].

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

Cell Viability Assay[2]

Cell Line:	Pancreatic cancer (SW1990, Aspc-1), breast cancer (mm-231, mm-468, MCF-7), liver cancer (Hep3B), cervical cancer (Hela), ovarian cancer (HOV-7, ES-2), normal cells (CHO, L929)
Concentration:	1-200 μM
Incubation Time:	24 hours
Result:	Inhibited the viability of cancer cells with higher GOT1 protein expression.

Apoptosis Analysis[2]

Cell Line:	ES-2 cells[2]
Concentration:	10, 20, 40 μM
Incubation Time:	24 hours
Result:	Showed a significant increase in apoptosis compared with the control group. Down regulated the expression of anti-apoptotic protein Bcl-2 and PARP.

Cell Cycle Analysis[3]

Cell Line:	LoVo or DLD1 cells
Concentration:	3, 10, 30 μM
Incubation Time:	6, 12, 24 hours
Result:	Caused cell cycle arrest in G1 phase in a dose- and time-dependent manner.

In Vivo

Adapalene (15-100 mg/kg; p.o. daily for 21 days) inhibits the growth of DLD1 cell-derived xenograft tumors in BALB/C nude mice[3].

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

Animal Model:	Female BALB/C nude mice (15 g, 4-5 weeks) were injected with DLD1 cells[3]
Dosage:	15, 20, 65, 100 mg/kg
Administration:	P.o. daily for 21 days
Result:	Significantly reduced tumor weight and volume.

CUSTOMER VALIDATION

- Proc Natl Acad Sci U S A. 2021 Jan 12;118(2):e2009539118.
- Eur J Pharmacol. 2019 May 15;851:174-185.

- Fundam Clin Pharmacol. 2020 Jun;34(3):380-388.
- Katedra farmakologie a toxikologie. 2020 Jul.

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REFERENCES

[1]. Shroot B, et, al. Pharmacology and chemistry of adapalene. J Am Acad Dermatol. 1997 Jun;36(6 Pt 2):S96-103.

[2]. Wang Q, et, al. Adapalene inhibits ovarian cancer ES-2 cells growth by targeting glutamic-oxaloacetic transaminase 1. Bioorg Chem. 2019 Dec;93:103315.

[3]. Shi XN, et, al. Adapalene inhibits the activity of cyclin-dependent kinase 2 in colorectal carcinoma. Mol Med Rep. 2015 Nov;12(5):6501-8.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA