Ro 41-5253

Cat. No.: HY-116248

CAS No.: 144092-31-9

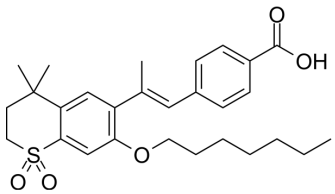
Molecular Formula: $C_{28}H_{36}O_5S$

Molecular Weight: 484.65

Target: RAR/RXR; Apoptosis

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

Storage: 4°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 : 100 mg/mL (206.33 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0633 mL	10.3167 mL	20.6334 mL
	5 mM	0.4127 mL	2.0633 mL	4.1267 mL
	10 mM	0.2063 mL	1.0317 mL	2.0633 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 (5.16 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.16 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ro 41-5253 is an orally active selective retinoic acid receptor alpha (RARα) antagonist. Ro 41-5253 can bind RARα without inducing transcription or affecting RAR/RXR heterodimerization and DNA binding. Ro 41-5253 can inhibit cancer cell proliferation and induce apoptosis, has antitumor activity[1][2].

IC_{50} & Target

IC50: 60 nM (RARα), 2.4 μM (RARβ), 3.3 μM (RARγ)[3].

In Vitro

Ro 41-5253 (1 nM-10 μM, 10 days) significantly inhibits MCF-7 and ZR 75.1 cell proliferation and induces cell apoptosis in a time and dose-dependent manner[1].

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

Cell Proliferation Assay[1]

Cell Line:	Human breast-carcinoma lines MCF-7 and ZR 75.1
Concentration:	1 nM-10 μM
Incubation Time:	10 days
Result:	Inhibited 81% MCF-7 cell growth at 10 μM, 30% cell growth at 1 μM and no significant inhibitory effect at concentrations below 0.1 μM. Inhibited 74% ZR 75.1 cell growth at 10 μM, 63% cell growth at 1 μM and 42% cell growth at 0.1 μM.

Apoptosis Analysis[1]

Cell Line:	Human breast-carcinoma lines MCF-7 and ZR 75.1
Concentration:	1 nM-10 μM
Incubation Time:	10 days
Result:	Induced 28.5, 21.6, 16 and 12% of MCF-7 cells apoptosis at 10 μM, 1 μM, 0.1 μM and 0.01 μM respectively on the fourth day while induced 58, 51, 36 and 21% of cells apoptosis at 10 μM, 1 μM, 0.1 μM and 0.01 μM respectively after six days. Induced 80, 65, 43 and 29% of ZR 75.1 cells apoptosis at 10 μM, 1 μM, 0.1 μM and 0.01 μM respectively on the sixth day.

In Vivo

Ro 41-5253 (oral gavage, 10-600 mg/kg, once a week, 4 weeks) can reduce tumor volume in female athymic Balb/mice transplanted with MCF-7 cell line[2].

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

Animal Model:	Six-week-old female athymic Balb/mice transplanted with MCF-7 cell line[2]
Dosage:	10, 30, 100, 300 and 600 mg/kg
Administration:	Oral gavage; once a week; 4 weeks
Result:	Resulted in a reduction in tumor volume at doses of 10, 30 and 100 mg/kg with no toxic side effects.

REFERENCES

[1]. S Toma, et al. RARalpha antagonist Ro 41-5253 inhibits proliferation and induces apoptosis in breast-cancer cell lines. Int J Cancer. 1998 Sep 25;78(1):86-94

[2]. Salvatore Toma, et al. Retinoids and human breast cancer: in vivo effects of an antagonist for RAR-alpha. Cancer Lett. 2005 Feb 28;219(1):27-31. doi: 10.1016/j.canlet.2004.06.018.

[3]. C Apfel, 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.

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