T0901317

Cat. No.: HY-10626

CAS No.: 293754-55-9

Molecular Formula: $C_{17}H_{12}F_9NO_3S$

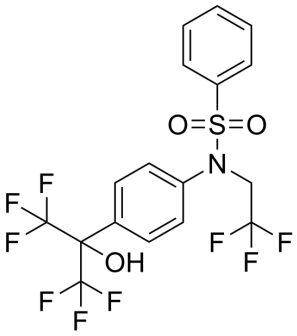
Molecular Weight: 481.33

Target: FXR; LXR; ROR; Apoptosis

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

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (207.76 mM; Need ultrasonic)
Ethanol : 100 mg/mL (207.76 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.0776 mL	10.3879 mL	20.7758 mL
5 mM	0.4155 mL	2.0776 mL	4.1552 mL
10 mM	0.2078 mL	1.0388 mL	2.0776 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: ≥ 3 mg/mL (6.23 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 3 mg/mL (6.23 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 3 mg/mL (6.23 mM); Clear solution
4. Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline
   Solubility: 2.5 mg/mL (5.19 mM); Suspended solution; Need ultrasonic
5. Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.19 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

T0901317 is an orally active and highly selective LXR agonist with an EC_{50} of 20 nM for LXRα[1]. T0901317 activates FXR with an EC_{50} of 5 μM[2]. T0901317 is RORα and RORγ dual inverse agonist with K_i values of 132 nM and 51 nM, respectively[3].

	T0901317 induces apoptosis and inhibits the development of atherosclerosis in low-density lipoprotein (LDL) receptor-deficient mice[4][5].
IC_{50} & Target	EC50: 20 nM (LXRα) and 5 μM (FXR)[1][2] Ki: 132 nM (RORα) and 51 nM (RORγ)[3]

In Vitro

rateand the T0901317 (5-50 μM; 72 hours) significantly inhibits cellular proliferation in CaOV3, SKOV3, A2780 (human ovarian carcinoma cell lines) in a dose-dependent and time-dependent manner[5].
T0901317 (10 μM; 24-72 hours) decreases the percentage of cells in S phase and increases the percentage of cells in the G0/G1 phase, indicating a cell cycle arrest at the G1-S checkpoint. The percentage of cells in G0/G1 phase increases in a time-dependent manner[5].
T0901317 (10-40 μM; 24 hours) results in a significant increase of cells in early apoptosis[5].
T0901317 (5-40 μM; 48 hours) results in an increase of p21 and p27 protein expression in a dose-dependent manner after 48 hours[5].

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

Cell Proliferation Assay[5]

Cell Line:	A2780, CaOV3 and SKOV3 ovarian cancer cell lines
Concentration:	5, 10, 20, 40 or 50 μM
Incubation Time:	72 hours
Result:	Inhibited cellular proliferation in all cell lines in a dose-dependent and time-dependent manner.

Cell Cycle Analysis[5]

Cell Line:	A2780, CaOV3 and SKOV3 cells
Concentration:	10 μM
Incubation Time:	24, 48 or 72 hours
Result:	Decreased the percentage of cells in S phase and increased the percentage of cells in the G0/G1 phase.

Apoptosis Analysis[5]

Cell Line:	CaOV3 cells
Concentration:	10 to 40 μM
Incubation Time:	24 hours
Result:	Resulted in a significant increase of cells in early apoptosis.

Western Blot Analysis[5]

Cell Line:	CaOV3 cells
Concentration:	5 to 40 μM
Incubation Time:	48 hours
Result:	Resulted in an increase of p21 and p27 protein expression in a dose-dependent manner.

In Vivo	T0901317 (10 mg/kg/day; orally; for 12 weeks) inhibits the progression of atherosclerosis[5].

T0901317 (i.p.; 50 mg/kg; twice weekly for 7 days) can protect male C57BL/6 mice from high fat diet-induced obesity and insulin resistance[6].

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

Animal Model:	8- to 10-week-old LDL receptor null mice[5]
Dosage:	10 mg/kg
Administration:	Orally; daily; for 12 weeks
Result:	Inhibited the progression of atherosclerosis.

CUSTOMER VALIDATION

- Nat Commun. 2024 Jul 21;15(1):6152.
- J Exp Clin Cancer Res. 2024 May 3;43(1):133.
- Cell Death Dis. 2024 Oct 18;15(10):754.
- Anal Chem. 2019 Jan 15;91(2):1501-1506.
- J Med Chem. 2024 Oct 15.

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REFERENCES

[1]. J R Schultz, et al. Role of LXRs in Control of Lipogenesis. Genes Dev. 2000 Nov 15;14(22):2831-8.

[2]. Rough JJ, et al. Anti-proliferative effect of LXR agonist T0901317 in ovarian carcinoma cells. J Ovarian Res. 2010 May 26;3:13.

[3]. Todd G Kirchgessner, et al. Beneficial and Adverse Effects of an LXR Agonist on Human Lipid and Lipoprotein Metabolism and Circulating Neutrophils. Cell Metab. 2016 Aug 9;24(2):223-33.

[4]. Keith A Houck, et al. T0901317 Is a Dual LXR/FXR Agonist. Mol Genet Metab. Sep-Oct 2004;83(1-2):184-7.

[5]. Naresh Kumar, et al. The Benzenesulfoamide T0901317 [N-(2,2,2-trifluoroethyl)-N-[4-[2,2,2-trifluoro-1-hydroxy-1-(trifluoromethyl)ethyl]phenyl]-benzenesulfonamide] Is a Novel Retinoic Acid Receptor-Related Orphan Receptor-Alpha/Gamma Inverse Agonist. Mol Pharmacol. 2010 Feb;77(2):228-36.

[6]. Mingming Gao, et al. The Liver X Receptor Agonist T0901317 Protects Mice From High Fat Diet-Induced Obesity and Insulin Resistance. AAPS J. 2013 Jan;15(1):258-66.

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