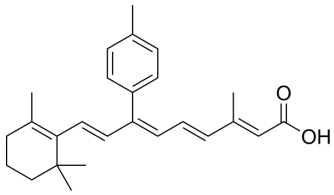
SR 11302

Cat. No.:	HY-15870
CAS No.:	160162-42-5
Molecular Formula:	$C_{26}H_{32}O_2$
Molecular Weight:	376.53
Target:	AP-1
Pathway:	Immunology/Inflammation
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (66.40 mM; ultrasonic and warming and heat to 80°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6558 mL	13.2792 mL	26.5583 mL
	5 mM	0.5312 mL	2.6558 mL	5.3117 mL
	10 mM	0.2656 mL	1.3279 mL	2.6558 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (6.64 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 1% CMC-Na/saline water
   Solubility: 2 mg/mL (5.31 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

SR 11302 is an activator protein-1 (AP-1) transcription factor inhibitor. SR 11302 is a retinoid that specifically inhibits AP-1 activity without activating the transcription of retinoic acid response element (RARE)[1].

IC_{50} & Target

AP-1[1]

In Vitro

SR 11302 (SR11302) show strong anti-AP-1 activity with selective binding with RARα and RARγ, but not with RARβ and RXRα [1].

SR 11302 (SR-11302; 1 μM) inhibits AP-1 transcription factor activity and decreases aldosterone levels by 61.9% in hypoxia-treated cells[2].

SR 11302 (SR-11302; 2 μM; 48 hours) inhibits Helicobacter pylori (H. pylori)-induced cell proliferation in adenocarcinoma gastric (AGS) cells[3].

	SR 11302 (2 μM; 24 hours) inhibits H. pylori-induced expression of β-catenin and c-myc in AGS cells[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	SR 11302 (SR11302; low dose 0.5 mg/kg and high dose 1 mg/kg body weight; orally gavaged daily) treatment reduces the total vascular lesion number and lesion size in Vldlr$^{-/-}$ mice in a dose-dependent manner[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Vldlr$^{-/-}$ mice[4]
Dosage:	Low dose 0.5 mg/kg and high dose 1 mg/kg body weight
Administration:	Orally gavaged daily from P5 to P15
Result:	High-dose from P5 to P15 reduced the total vascular lesion number by 48% and decreased the lesion size by 40%, without detectable signs of toxicity in mice, including no change in body weight.

CUSTOMER VALIDATION

- Cell Mol Immunol. 2022 May 12.
- J Agric Food Chem. 2022 Feb 16;70(6):1996-2009.
- Viruses. 2022, 14(7), 1485.
- Vet Microbiol. 2021, 109061.

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REFERENCES

[1]. C Huang, et al. Blocking activator protein-1 activity, but not activating retinoic acid response element, is required for the antitumor promotion effect of retinoic acid. Proc Natl Acad Sci U S A. 1997 May 27;94(11):5826-30.

[2]. Bradley A Maron, et al. Upregulation of steroidogenic acute regulatory protein by hypoxia stimulates aldosterone synthesis in pulmonary artery endothelial cells to promote pulmonary vascular fibrosis. Circulation. 2014 Jul 8;130(2):168-79.

[3]. Eunyoung Byun, et al. Activation of NF-κB and AP-1 Mediates Hyperproliferation by Inducing β-Catenin and c-Myc in Helicobacter pylori-Infected Gastric Epithelial Cells. Yonsei Med J. 2016 May;57(3):647-51.

[4]. Ye Sun, et al. Inflammatory signals from photoreceptor modulate pathological retinal angiogenesis via c-Fos. J Exp Med. 2017 Jun 5;214(6):1753-1767.

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