SC-236

Cat. No.: HY-W010983

CAS No.: 170569-86-5

Molecular Formula: $C_{16}H_{11}ClF_3N_3O_2S$

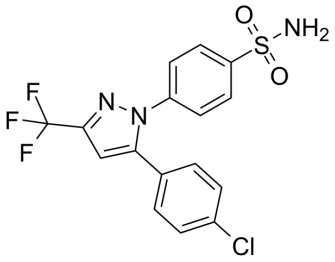
Molecular Weight: 401.79

Target: COX; PPAR; Apoptosis

Pathway: Immunology/Inflammation; Cell Cycle/DNA Damage; Apoptosis

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (248.89 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.4889 mL	12.4443 mL	24.8886 mL
	5 mM	0.4978 mL	2.4889 mL	4.9777 mL
	10 mM	0.2489 mL	1.2444 mL	2.4889 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 (6.22 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.22 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.22 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

SC-236 is an orally active COX-2 specific inhibitor (IC_{50} = 10 nM) and a PPARγ agonist. SC-236 suppresses activator protein-1 (AP-1) through c-Jun NH2-terminal kinase. SC-236 exerts anti-inflammatory effects by suppressing phosphorylation of ERK in a murine model[1][2][3][4][5].

IC_{50} & Target

COX-2	COX-1
10 nM (IC_{50})	17.8 μM (IC_{50})

In Vitro

SC-236 (15 μM, 30 min) suppresses the side effects of NSAIDs and prevented inflammation in vECs subjected to ALSS[1].

SC-236 significantly induces PPARγ expression in HSCs and acted as a potent PPARγ agonist in a luciferase-reporter trans-activation assay[2].
SC-236 strongly inhibits, in a time- and concentration-dependent manner, macrophageviability[2].
SC-236, either alone or in combination with 15d-PGJ2, induced a marked pro-apoptotic effect in HSCs in culture[2].
SC-236 mediates antitumor effect by modulation of AP-1-signaling pathway[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	vECs.
Concentration:	15 μM
Incubation Time:	30 min.
Result:	Showd significant reduction in COX-2 level and increase in IκBα level, thus preventing ALSS-induced NFκB activation and inflammation in vECs.

Western Blot Analysis[2]

Cell Line:	COS 7 cells.
Concentration:	3 and 10 μM.
Incubation Time:	18 h (combined with 15d-PGJ_2).
Result:	Acted in a concentration-dependent manner as a PPARγ agonist.

In Vivo

SC-236 (6 mg/kg, gavage) exhibits anti-fibrotic properties in CCl4- treated animals[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Seventy-six male adult Wistar rats weighing 200-220 g (CCl_4-treated)[2].
Dosage:	6 mg/kg.
Administration:	Orally, 3 times per week.
Result:	A marked induction of COX-2 protein expression was detected by immunohistochemistry in the liver of CCl4-treated rats. Significantly reduced the degree of liver fibrosis. Dramatically suppressed α-SMA expression in CCl4-treated rats.

REFERENCES

[1]. Shao-Yu Fang, et al. Reduction in MicroRNA-4488 Expression Induces NFκB Translocation in Venous Endothelial Cells Under Arterial Flow. Cardiovasc Drugs Ther. 2020 Sep 9.

[2]. Anna Planagumà, et al. The selective cyclooxygenase-2 inhibitor SC-236 reduces liver fibrosis by mechanisms involving non-parenchymal cell apoptosis and PPARgamma activation. FASEB J. 2005 Jul;19(9):1120-2.

[3]. Benjamin Chun-Yu Wong, et al. Cyclooxygenase-2 inhibitor (SC-236) suppresses activator protein-1 through c-Jun NH2-terminal kinase. Gastroenterology. 2004 Jan;126(1):136-47.

[4]. Su-Jin Kim, et al. The COX-2 inhibitor SC-236 exerts anti-inflammatory effects by suppressing phosphorylation of ERK in a murine model. Life Sci. 2007 Aug 23;81(11):863-72.

[5]. T D Penning, et al. Synthesis and biological evaluation of the 1,5-diarylpyrazole class of cyclooxygenase-2 inhibitors: identification of 4-[5-(4-methylphenyl)-3-(trifluoromethyl)-1H-pyrazol-1-yl]benze nesulfonamide (SC-58635, celecoxib). J Med Chem. 1997 Apr 25;40(9):1347-65.

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