RS102895 hydrochloride

Cat. No.: HY-18611

CAS No.: 1173022-16-6

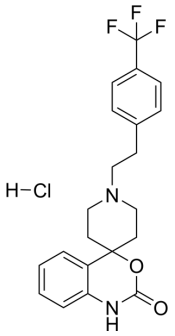
Molecular Formula: $C_{21}H_{22}ClF_3N_2O_2$

Molecular Weight: 426.86

Target: CCR

Pathway: GPCR/G Protein; Immunology/Inflammation

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 33.33 mg/mL (78.08 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.3427 mL	11.7134 mL	23.4269 mL
5 mM	0.4685 mL	2.3427 mL	4.6854 mL
10 mM	0.2343 mL	1.1713 mL	2.3427 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 (5.86 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.86 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.86 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

RS102895 hydrochloride is a potent CCR2 antagonist, with an IC_{50} of 360 nM, and shows no effect on CCR1.

IC_{50} & Target

CCR2 360 nM (IC_{50})	CCR1 17800 nM (IC_{50})	Human α_{1a} receptor 130 nM (IC_{50})	Human α_{1d} receptor 320 nM (IC_{50})
5HT-1a receptor 470 nM (IC_{50})			

In Vitro	RS102895 hydrochloride is a potent CCR2 antagonist, with an IC_{50} of 360 nM, and shows no effect on CCR1. RS102895 also inhibits human α1a and α1d receptors, rat brain cortex 5HT1a receptor in cells with IC_{50}s of 130, 320, 470 nM, respectively. RS102895 suppresses wild type and D284N mutant MCP-1 receptor (IC_{50}, 550 nM and 568 nM, respectively), less potently inhibits D284A MCP-1 receptor (IC_{50}, 1892 nM), and has no effects on E291A, E291Q, D284A/E291A or D284N/E291Q (IC_{50}, >100,000?nM)[1]. RS102895 ameliorates the increased extracellular matrix (ECM) protein expression by inhibition of CCR2 at 10 μM, and obviously blocks fibronectin and type IV collagen protein expression in high glucose (HG)-stimulated mesangial cells (MCs) at 1 or 10 μM. RS102895 (10 μM) also abrogates the increased TGF-1 levels in MCs treated with MCP-1[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	RS102895 (3 g/L) causes progressive decrease in pain threshold in rats with bone cancer pain (BCP) at day 3-9 after surgery via intrathecal injection, but the pain threshold increases after 12 days. RS102895 also potently reverses the pattern of NR2B, nNOS, and SIGIRR expression in spinal cord[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Transfected mesangial cells (MCs) are serum restricted for 24 h, after which the medium is replaced by serum-free DMEM containing normal glucose (NG; 5.6 mM), NG+Mannitol (NG+M; 24.4 mM), or high glucose (HG; 30 mM). In addition, nontransfected MCs are cultured under NG, NG+M, or HG with or without RS102895 or anti-TGF-1 antibody (25 μg/mL). Nontransfected MCs are also exposed to medium containing recombinant human MCP-1 (10 ng/mL) or recombinant human TGF-1 (2 ng/mL). At 24 h after the media change, cells are harvested and the conditioned culture media are collected[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Rats[3] CCR2 antagonist RS102895 is dissolved in DMSO. Rats receive daily intrathecal injection of either RS102895 (3 g/L) 10 μL or 10 % DMSO 10 μL between 9 and 20 days after operation. All rats are randomLy divided into five groups (n = 10 per group): Sham group, Sham + RS102895 group, BCP group, BCP + RS102895 group, and BCP + DMSO group. Rats are sacrificed 20 days after operation and the tissue samples from the L4-L5 spinal cord segments are rapidly removed and immediately frozen in liquid nitrogen and stored at −80°C until use for RT-PCR and Western blot[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Sci Adv. 2023 Aug 2;9(31):eadg6856.
- Theranostics. 2020 Apr 27;10(13):5687-5703
- J Adv Res. 2020 Jul 22;28:231-243.
- J Neuroinflammation. 2023 Nov 17;20(1):270.
- J Eur Acad Dermatol Venereol. 2020 Apr;34(4):862-872.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Mirzadegan T, et al. Identification of the binding site for a novel class of CCR2b chemokine receptor antagonists: binding to a common chemokine receptor motif within the helical bundle. J Biol Chem. 2000 Aug 18;275(33):25562-71.

[2]. Park J, et al. MCP-1/CCR2 system is involved in high glucose-induced fibronectin and type IV collagen expression in cultured mesangial cells. Am J Physiol Renal Physiol. 2008 Sep;295(3):F749-57.

[3]. Ren F, et al. Analgesic Effect of Intrathecal Administration of Chemokine Receptor CCR2 Antagonist is Related to Change in Spinal NR2B, nNOS, and SIGIRR Expression in Rat with Bone Cancer Pain. Cell Biochem Biophys. 2015 Jun;72(2):611-6.

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