J-113863

Cat. No.: HY-103360

CAS No.: 353791-85-2

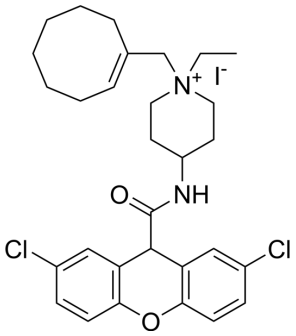
Molecular Formula: $C_{30}H_{37}Cl_2IN_2O_2$

Molecular Weight: 655.44

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 : 50 mg/mL (76.28 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.5257 mL	7.6285 mL	15.2569 mL
	5 mM	0.3051 mL	1.5257 mL	3.0514 mL
	10 mM	0.1526 mL	0.7628 mL	1.5257 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 (3.81 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (3.81 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

J-113863 is a potent and selective CCR1 antagonist with IC_{50} values of 0.9 nM and 5.8 nM for human and mouse CCR1 receptors, respectively. J-113863 is also a potent antagonist of the human CCR3 (IC_{50} of 0.58 nM) , but a weak antagonist of the mouse CCR3 (IC_{50} of 460 nM). J-113863 is inactive against CCR2, CCR4 and CCR5, as well as the LTB4 or TNF-α receptors. Anti-inflammatory effect[1][2][3].

IC_{50} & Target				
	CCR1 0.9 nM (IC_{50}, Human CCR1)	CCR1 5.8 nM (IC_{50}, Mouse CCR1)	CCR3 0.58 nM (IC_{50}, Human CCR3)	CCR3 460 nM (IC_{50}, Mouse CCR3)

In Vitro

Modified Vaccinia virus Ankara (MVA) but not MVA and vaccinia virus (VACV) infected MH-S cells increase the expression of the CXCR2 acting chemokine CXCL2. MH-S cells constitutively produce CCL2 and CCR1 acting chemokines CCL3, CCL5 and

CCL9. Consequently, supernatants of mock treated and virus infected MH-S cells induce chemotaxis of murine promyelocyte MPRO cells and human monocytic THP-1 cells at the same level. However, supernatants of MVA infected MH-S cells significantly increase chemotaxis of the CCR2 deficient human monocytic cell line U-937. Chemotaxis of all above cell types is inhibited by J-113863[1].

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

In Vivo

J-113863 (3-10 mg/kg; intraperitoneal injection; once daily; for 11 days; DBA-1 male mice) treatment improves paw inflammation and joint damage, and dramatically decreases cell infiltration into joints in arthritic mice[2].

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

Animal Model:	DBA-1 male mice (10-12 weeks) induced with Collagen[2]
Dosage:	3 mg/kg, 10 mg/kg
Administration:	Intraperitoneal injection; once daily; for 11 days
Result:	Improved paw inflammation and joint damage, and dramatically decreased cell infiltration into joints.

CUSTOMER VALIDATION

- Bioact Mater. 2021 Jan 7;6(7):2039-2057.
- Inflamm Regen. 2023 Mar 3;43(1):18.
- J Ethnopharmacol. 2022 May 10;289:115051.

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REFERENCES

[1]. Lehmann MH, et al. Modified Vaccinia virus Ankara but not vaccinia virus induces chemokine expression in cells of the monocyte/macrophage lineage. Virol J. 2015 Feb 12;12:21.

[2]. Amat M, et al. Pharmacological blockade of CCR1 ameliorates murine arthritis and alters cytokine networks in vivo. Br J Pharmacol. 2006 Nov;149(6):666-75.

[3]. Naya A, et al. Design, synthesis, and discovery of a novel CCR1 antagonist. J Med Chem. 2001 Apr 26;44(9):1429-35.

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