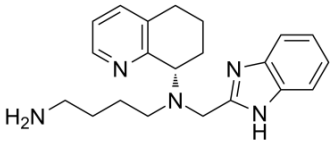
Mavorixafor

Cat. No.:	HY-50101
CAS No.:	558447-26-0
Molecular Formula:	$C_{21}H_{27}N_5$
Molecular Weight:	349.47
Target:	CXCR; HIV
Pathway:	GPCR/G Protein; Immunology/Inflammation; Anti-infection
Storage:	4°C, stored under nitrogen * In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



BIOLOGICAL ACTIVITY

Description	Mavorixafor (AMD-070) is a potent, selective and orally available CXCR4 antagonist, with an IC_{50} value of 13 nM against CXCR4 ^{125}I-SDF binding, and also inhibits the replication of T-tropic HIV-1 (NL4.3 strain) in MT-4 cells and PBMCs with an IC_{50} of 1 and 9 nM, respectively.
IC_{50} & Target	^{125}I-SDF-CXCR4 13 nM (IC_{50}) HIV-1 (NL4.3 strain) 1 nM (IC_{50}, in MT-4 cells) HIV-1 (NL4.3 strain) 9 nM (IC_{50}, in PBMCs) HIV-1 (NL4.3 strain) 3 nM (IC90, in MT-4 cells) HIV-1 (NL4.3 strain) 26 nM (IC90, in PBMCs)
In Vitro	Mavorixafor (AMD-070) is a potent and orally available CXCR4 antagonist, with an IC_{50} value of 13 nM against CXCR4 ^{125}I-SDF binding, and also inhibits the replication of T-tropic HIV-1 (NL4.3 strain) in MT-4 cells and PBMCs with an IC_{50} of 1 and 9 nM, respectively. Mavorixafor (AMD-070) shows no effect on other chemokine receptors (CCR1, CCR2b, CCR4, CCR5, CXCR1, and CXCR2)[1]. Mavorixafor (AMD-070) (6.6 μM) significantly suppresses the anchorage-dependent growth, the migration and matrigel invasion of the B88-SDF-1 cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Mavorixafor (AMD-070) (2 mg/kg, p.o.) significantly reduces the number of metastatic lung nodules in mice, and lowers the expression of human Alu DNA in mice, without body weight loss[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Cells are seeded on a 96-well plate at 5×10^3 cells/well in DMEM containing 10% FCS. Twenty-four hours later, the cells are treated with or without 2 μM AMD3100 or 6.6 μM AMD-070. After 24 or 48 h, the number of cells is quantified by an assay using MTT[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] BALB/c nude mice are maintained under pathogen-free conditions. The experiments are initiated when the mice are 8 weeks of age. Briefly, the cells are inoculated into the blood vessels of nude mice (1×10^6). These mice are sacrificed at day

	49. The presence or absence of distant metastases is confirmed by hematoxylin and eosin (H&E) staining. For experimental chemotherapy, the mice are treated by the daily oral administration of 0.2 mL of saline for a vehicle or the same volume of Mavorixafor (AMD-070) (2 mg/kg)[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- PLoS One. 2016 Mar 21;11(3):e0151765.

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REFERENCES

[1]. Skerlj RT, et al. Discovery of novel small molecule orally bioavailable C-X-C chemokine receptor 4 antagonists that are potent inhibitors of T-tropic (X4) HIV-1 replication. J Med Chem. 2010 Apr 22;53(8):3376-88.

[2]. Uchida D, et al. Effect of a novel orally bioavailable CXCR4 inhibitor, AMD070, on the metastasis of oral cancer cells. Oncol Rep. 2018 Jul;40(1):303-308.

[3]. Chow LN, et al. Impact of a CXCL12/CXCR4 Antagonist in Bleomycin (BLM) Induced Pulmonary Fibrosis and Carbon Tetrachloride (CCl4) Induced Hepatic Fibrosis in Mice. PLoS One. 2016 Mar 21;11(3):e0151765.

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