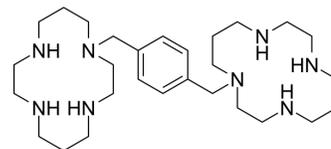


Plerixafor

Cat. No.:	HY-10046		
CAS No.:	110078-46-1		
Molecular Formula:	C ₂₈ H ₅₄ N ₈		
Molecular Weight:	502.78		
Target:	CXCR; HIV		
Pathway:	GPCR/G Protein; Immunology/Inflammation; Anti-infection		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

Ethanol : 50 mg/mL (99.45 mM; Need ultrasonic)
 DMSO : 1.96 mg/mL (3.90 mM; ultrasonic and warming and adjust pH to 5 with HCl and heat to 60°C)
 H₂O : < 0.1 mg/mL (ultrasonic) (insoluble)

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	1.9889 mL	9.9447 mL	19.8894 mL
	5 mM	0.3978 mL	1.9889 mL	3.9779 mL
	10 mM	0.1989 mL	0.9945 mL	1.9889 mL

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

In Vivo

- Add each solvent one by one: 10% EtOH >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 3 mg/mL (5.97 mM); Clear solution
- Add each solvent one by one: 10% EtOH >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 3 mg/mL (5.97 mM); Clear solution
- Add each solvent one by one: 10% EtOH >> 90% corn oil
Solubility: ≥ 3 mg/mL (5.97 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Plerixafor (AMD 3100) is a selective CXCR4 antagonist with an IC₅₀ of 44 nM. Plerixafor, an immunostimulant and a hematopoietic stem cell (HSC) mobilizer, is an allosteric agonist of CXCR7. Plerixafor inhibits HIV-1 and HIV-2 replication with an EC₅₀ of 1-10 nM^{[1][2][3][4][7]}.

IC₅₀ & Target

¹²⁵I-CXCL12-CXCR4

¹²⁵I-CXCL12-CXCR7

HIV-1

HIV-2

	44 nM (IC ₅₀)	1-10 nM (EC ₅₀)	1-10 nM (EC ₅₀)
In Vitro	<p>The CXCR4 inhibitor Plerixafor (AMD3100) is a potent inhibitor of CXCL12-mediated chemotaxis (IC₅₀, 5.7 nM) with a potency slightly better than its affinity for CXCR4. Plerixafor interferes with the interaction of CXCR4 with its natural ligand, SDF-1 (CXCL12). Treating the cells with CX771 or CXCL11 has no effect on CXCL12-mediated MOLT-4 or U937 TEM. In contrast, 10 μM Plerixafor inhibits CXCL12-mediated TEM in both cells lines^[1].</p> <p>Plerixafor prevents the infiltration of tumor-associated macrophages (TAMs) into the tumor tissues^[8]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>		
In Vivo	<p>Plerixafor (2 mg/kg) administration to UUO mice exacerbates renal interstitial T cell infiltration, resulting in increased production of the pro-inflammatory cytokines IL-6 and IFN-γ and decreased expression of the anti-inflammatory cytokine IL-10^[5].</p> <p>Both perivascular and interstitial fibrosis are significantly reduced by the CXCR4 antagonist, Plerixafor (AMD3100) at 8 weeks^[6]. LD50, mouse, SC: 16.3 mg/kg; LD50, rat, SC: >50 mg/kg; LD50, mouse and rat, IV injection: 5.2 mg/kg.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>		

PROTOCOL

Cell Assay ^[2]

U87MG cells are seeded in 96-well plates at the density of 6×10³ cells in 200 μL/well and treated with CXCL12, Plerixafor or with peptide R. MTT (5 μg/mL) is added at each time point (24, 48, 72 h) during the final 2 h of treatment. After removing cell medium, 100 μL DMSO are added and optical densities measured at 595 nm with a LT-4000MS Microplate Reader. Measurements are made in triplicates from three independent experiments^[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Administration ^{[3][4]}

Mice^[3]
 Male C57bl/6 mice (6-7 weeks old, weighing 20 g) are used. The animals are acclimated to the housing environment, which is SPF and had a temperature of 22°C and a 12h/12h light/dark cycle for a week. Then, they are randomly divided into following experimental groups, with 8 mice in each group: normal (no specific intervention), UUO+AMD3100 (mice received UUO surgery and 2 mg/kg AMD3100), and UUO+PBS (mice received UUO surgery and the same volume of PBS). AMD3100 and PBS are administered via intraperitoneal injection every day until sacrifice.

Rats^[4]
 The CXCR4 antagonist, AMD3100 dissolved in H₂O, is delivered in the type 2 diabetic sand rat model at a dose of 6 mg/kg per day for 8 weeks. In complementary studies, the effect of CXCR4 antagonism (AMD3100 6mg/kg/d) on regulatory T cell numbers is examined. For these studies, AMD3100 or vehicle is delivered via minipump for a period of one week. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Immunity. 2024 Feb 13;57(2):364-378.e9.
- Cell Mol Immunol. 2020 Mar;17(3):283-299.
- Adv Funct Mater. 2020, 2000309.
- Bioact Mater. 2021 Jan 7;6(7):2039-2057.
- Nano Today. 2022, 47: 101689.

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- [2]. Mercurio L, et al. Targeting CXCR4 by a selective peptide antagonist modulates tumor microenvironment and microglia reactivity in a human glioblastoma model. *J Exp Clin Cancer Res.* 2016 Mar 25;35:55.
- [3]. Yang J, et al. Continuous AMD3100 Treatment Worsens Renal Fibrosis through Regulation of Bone Marrow Derived Pro-Angiogenic Cells Homing and T-Cell-Related Inflammation. *PLoS One.* 2016 Feb 22;11(2):e0149926.
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- [5]. De Clercq E, et al. Mozobil® (Plerixafor, AMD3100), 10 years after its approval by the US Food and Drug Administration. *Antivir Chem Chemother.* 2019 Jan-Dec;27:2040206619829382.
- [6]. Seki JT, et al. Chemical Stability of Plerixafor after Opening of Single-Use Vial. *Can J Hosp Pharm.* 2017 Jul-Aug;70(4):270-275.
- [7]. Schols D, et al. HIV co-receptor inhibitors as novel class of anti-HIV drugs. *Antiviral Res.* 2006 Sep;71(2-3):216-26.
- [8]. Zheng J, et al. Toward Normalization of the Tumor Microenvironment for Cancer Therapy. *Integr Cancer Ther.* 2019;18:1534735419862352.
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Caution: Product has not been fully validated for medical applications. For research use only.

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