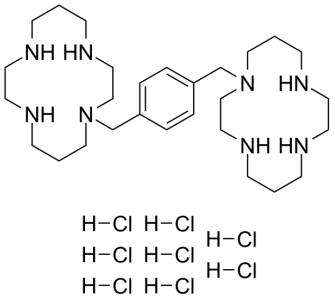
Plerixafor octahydrochloride

Cat. No.:	HY-50912
CAS No.:	155148-31-5
Molecular Formula:	$C_{28}H_{62}Cl_8N_8$
Molecular Weight:	794.47
Target:	CXCR; Virus Protease; HIV
Pathway:	GPCR/G Protein; Immunology/Inflammation; Anti-infection
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

H_2O : 100 mg/mL (125.87 mM; Need ultrasonic)
DMSO : < 1 mg/mL (insoluble or slightly soluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.2587 mL	6.2935 mL	12.5870 mL
5 mM	0.2517 mL	1.2587 mL	2.5174 mL
10 mM	0.1259 mL	0.6294 mL	1.2587 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 120 mg/mL (151.04 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Plerixafor octahydrochloride (AMD3100 octahydrochloride) is a selective CXCR4 antagonist with an IC_{50} of 44 nM.

IC_{50} & Target

^{125}I-CXCL12-CXCR4	^{125}I-CXCL12-CXCR7	HIV-1	HIV-2
44 nM (IC_{50})		1-10 nM (EC50)	1-10 nM (EC50)

In Vitro

The CXCR4 inhibitor Plerixafor (AMD3100) is a potent inhibitor of CXCL12-mediated chemotaxis (IC_{50}, 5.7 nM) with a potency slightly better than its affinity for CXCR4. Treating the cells with CCX771 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]. Plerixafor (10 μM)-treated cells show a moderate reduction in cell proliferation compared to CXCL12-stimulated cells, which do not reach statistical significance[2].

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

In Vivo

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[3]. Both perivascular and interstitial fibrosis are significantly reduced by the CXCR4 antagonist, Plerixafor (AMD3100) at 8 weeks[4]. LD50, mouse, SC: 16.3 mg/kg; LD50, rat, SC: >50 mg/kg; LD50, mouse and rat, IV injection: 5.2 mg/kg. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	U87MG cells are seeded in 96-well plates at the density of 6×10^3 cells in 200 μL/well and treated with CXCL12, Plerixafor or with peptide R, as described in the previous "Treatments" section. 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 H_2O, 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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REFERENCES

[1]. Zabel BA, et al. Elucidation of CXCR7-mediated signaling events and inhibition of CXCR4-mediated tumor cell transendothelial migration by CXCR7 ligands. J Immunol. 2009 Sep 1;183(5):3204-11.

[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.

[4]. Chu PY, et al. CXCR4 Antagonism Attenuates the Development of Diabetic Cardiac Fibrosis. PLoS One. 2015 Jul 27;10(7):e0133616.

[5]. Schols D, et al. HIV co-receptor inhibitors as novel class of anti-HIV drugs. Antiviral Res. 2006 Sep;71(2-3):216-26.

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