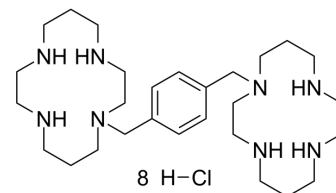


## Plerixafor octahydrochloride

<b>Cat. No.:</b>	HY-50912
<b>CAS No.:</b>	155148-31-5
<b>Molecular Formula:</b>	C <sub>28</sub> H <sub>54</sub> N <sub>8</sub> ·8HCl
<b>Molecular Weight:</b>	794.47
<b>Target:</b>	CXCR; Virus Protease; HIV
<b>Pathway:</b>	GPCR/G Protein; Immunology/Inflammation; Anti-infection
<b>Storage:</b>	4°C, sealed storage, away from moisture * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



### SOLVENT & SOLUBILITY

<b>In Vitro</b>	H <sub>2</sub> O : 100 mg/mL (125.87 mM; Need ultrasonic)					
	DMSO : 1 mg/mL (1.26 mM; ultrasonic and warming and heat to 80°C)					
	<b>Preparing Stock Solutions</b>	<b>Solvent</b>	<b>Mass</b>	<b>1 mg</b>	<b>5 mg</b>	<b>10 mg</b>
		<b>Concentration</b>				
		<b>1 mM</b>		1.2587 mL	6.2935 mL	12.5870 mL
<b>5 mM</b>			0.2517 mL	1.2587 mL	2.5174 mL	
	<b>10 mM</b>		0.1259 mL	0.6294 mL	1.2587 mL	
Please refer to the solubility information to select the appropriate solvent.						
<b>In Vivo</b>	1. Add each solvent one by one: PBS Solubility: 120 mg/mL (151.04 mM); Clear solution; Need ultrasonic					

### BIOLOGICAL ACTIVITY

<b>Description</b>	Plerixafor octahydrochloride (AMD3100 octahydrochloride) is a selective CXCR4 antagonist with an IC <sub>50</sub> of 44 nM.			
<b>IC<sub>50</sub> &amp; Target</b>	<sup>125</sup> I-CXCL12-CXCR4 44 nM (IC <sub>50</sub> )	<sup>125</sup> I-CXCL12-CXCR7	HIV-1 1-10 nM (EC <sub>50</sub> )	HIV-2 1-10 nM (EC <sub>50</sub> )
<b>In Vitro</b>	The CXCR4 inhibitor Plerixafor (AMD3100) is a potent inhibitor of CXCL12-mediated chemotaxis (IC <sub>50</sub> , 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 <sup>[1]</sup> . Plerixafor (10 μM)-treated cells show a moderate reduction in cell proliferation compared to CXCL12-stimulated cells, which do not reach statistical significance <sup>[2]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.			
<b>In Vivo</b>	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- $\gamma$  and decreased expression of the anti-inflammatory cytokine IL-10<sup>[3]</sup>. Both perivascular and interstitial fibrosis are significantly reduced by the CXCR4 antagonist, Plerixafor (AMD3100) at 8 weeks<sup>[4]</sup>. 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 <sup>[2]</sup>

U87MG cells are seeded in 96-well plates at the density of  $6 \times 10^3$  cells in 200  $\mu$ L/well and treated with CXCL12, Plerixafor or with peptide R, as described in the previous "Treatments" section. MTT (5  $\mu$ g/mL) is added at each time point (24, 48, 72 h) during the final 2 h of treatment. After removing cell medium, 100  $\mu$ L 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<sup>[2]</sup>. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

### Animal Administration <sup>[3][4]</sup>

#### Mice<sup>[3]</sup>

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<sup>[4]</sup>

The CXCR4 antagonist, AMD3100 dissolved in H<sub>2</sub>O, 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.

**Caution: Product has not been fully validated for medical applications. For research use only.**

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