

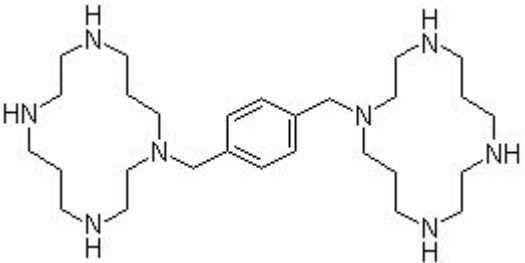


## Product Introduction

### Plerixafor (AMD3100)

Plerixafor is a chemokine receptor antagonist for CXCR4 and CXCL12-mediated chemotaxis with IC<sub>50</sub> of 44 nM and 5.7 nM, respectively.

#### Technical Data:

<b>Molecular Weight (MW):</b>	502.78	
<b>Formula:</b>	C <sub>28</sub> H <sub>54</sub> N <sub>8</sub>	
<b>Solubility (25°C)</b>	DMSO <1 mg/mL	
<b>* &lt;1 mg/ml means slightly soluble or insoluble:</b>	Water 6 mg/mL	
	Ethanol 100 mg/mL	
<b>Purity:</b>	>98%	
<b>Storage:</b>	3 years -20°C Powder 6 months -80°C in DMSO	
<b>CAS No.:</b>	110078-46-1	

#### Biological Activity

Plerixafor inhibits CXCL12-mediated chemotaxis with a potency slightly better than its affinity for CXCR4. [1] Plerixafor also antagonizes SDF-1/CXCL12 ligand binding with an IC<sub>50</sub> of 651 nM. Plerixafor inhibits SDF-1 mediated GTP-binding, SDF-1 mediated calcium flux and SDF-1 stimulated chemotaxis with IC<sub>50</sub> of 27 nM, 572 nM and 51 nM, respectively. Plerixafor does not inhibit calcium flux against cells expressing CXCR3, CCR1, CCR2b, CCR4, CCR5 or CCR7 when stimulated with their cognate ligands, nor does Plerixafor inhibit receptor binding of LTB<sub>4</sub>. Plerixafor does not, on its own, induce a calcium flux in the CCRF-CEM cells,

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which express multiple GPCRs including CXCR4, CCR4 and CCR7. [2]

A single topical application of Plerixafor promotes wound healing in diabetic mice by increasing cytokine production, mobilizing bone marrow EPCs, and enhancing the activity of fibroblasts and monocytes/macrophages, thereby increasing both angiogenesis and vasculogenesis. [3] Cohorts of mice are administered with PBS, IGF1, PDGF, SCF, or VEGF for five consecutive days and Plerixafor on the 5th day. The number and size of the colonies are highest in IGF1 plus Plerixafor injected mice compared to PDGF, SCF and VEGF treated groups, in combination with Plerixafor. [4]

## References

- [1] Zabel BA, et al. J Immunol. 2009, 183(5), 3204-3211.
- [2] Fricker SP, et al. Biochem Pharmacol. 2006, 72(5), 588-596.
- [3] Nishimura Y, et al. J Invest Dermatol. 2012, 132(3 Pt 1), 711-720.
- [4] Kumar S, et al. Bone. 2012, 50(4), 1012-1018.



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