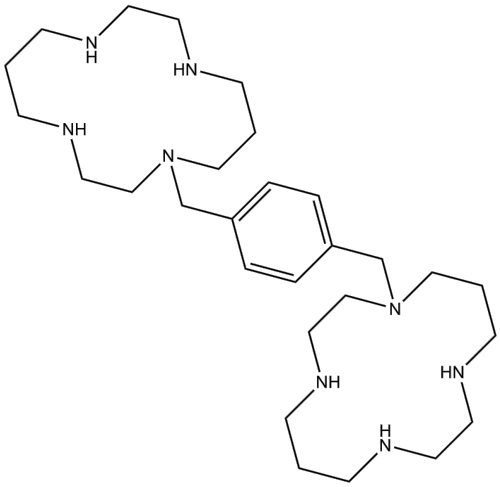
Product Data Sheet

Product Name: Plerixafor (AMD3100)
Cat. No.: GC14745

Chemical Properties

Cas. No.	110078-46-1		
Chemical Name	1-[[4-(1,4,8,11-tetrazacyclotetradec-1-ylmethyl)phenyl]methyl]-1,4,8,11-tetrazacyclotetradecane		
SMILES	C1CNCCNCCCN(CCNC1)CC2=CC=C(C=C2)CN3CCCNCCNCCCNCC3		
Formula	$C_{28}H_{54}N_8$	M.Wt	502.78
Solubility	≥ 25.1mg/mL in Ethanol	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

Cell experiment [1]:	
Cell lines	LoVo cells (human colon cancer cell line)

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Preparation Method	LoVo cells were maintained in DMEM supplemented with 10% FBS at 37°C, 5% CO_2. LoVo cells were treated with Plerixafor at 1μg/mL in the presence of 20ng/mL G-CSF for 24h, after which cell viability, proliferation, migration and invasion were assessed, colony formation and Transwell assays, and CXCR4, E-cadherin, Vimentin, Fibronectin and ZEB1 expression were measured.
Reaction Conditions	1μg/ml; 24h
Applications	Plerixafor reduced CXCR4 expression in LoVo cells. Plerixafor suppressed LoVo cell viability, proliferation, migration and invasion. Plerixafor increased E-cadherin expression and decreased Vimentin, Fibronectin and ZEB1 expression in LoVo cells.
Animal experiment [2]:	
Animal models	Male BALB/c nude mice
Preparation Method	Mice were anesthetized and inoculated with 1×10^6 DU 145 or PC-3 prostate cancer cells expressing KLF5K369Q (KLF5KQ) into the tibia (20μL per tibia). Starting at day 11 after tumor inoculation, mice were intraperitoneally administered Plerixafor at 3.5mg/kg/day alone or in combination with docetaxel (10mg/kg, i.p., twice a week). At 38 days (DU 145) or 43 days (PC-3) after inoculation, mice were euthanized and tibias were collected for X-ray radiography, H&E staining, and TRAP staining to evaluate bone lesion scores, tumor area ratios, and TRAP-positive osteoclast occurrence at the tumor-bone interface.
Dosage form	3.5mg/kg/day; i.p.; daily for 28 or 32 days

Caution: Product has not been fully validated for medical applications. For research use only.
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Applications	Plerixafor administration suppressed KLF5KQ-induced bone metastatic tumor growth and reduced tumor area in the tibia. Plerixafor decreased TRAP-positive osteoclast occurrence at the tumor-bone interface in KLF5KQ tibial tumors. Combined treatment with Plerixafor and docetaxel produced a synergistic suppressive effect on KLF5KQ-induced bone metastatic lesions, with combination index values of 0.59 for PC-3 and 0.40 for DU 145 tumors. Plerixafor alone selectively inhibited IL-11 expression in KLF5KQ tibial tumors.

References:

[1] Ma G, Jiang J, He T, et al. G-CSF promotes H3K27ac-modified KLF5 to activate CXCR4 expression and drive colon cancer growth and metastasis. Eur J Med Res. 2026;31(1):207.

[2] Zhang B, Li Y, Wu Q, et al. Acetylation of KLF5 maintains EMT and tumorigenicity to cause chemoresistant bone metastasis in prostate cancer. Nat Commun. 2021 Mar 17;12:1714.

Background

Plerixafor (AMD3100) is a specific small-molecule CXCR4 antagonist (IC_{50}=44nM), an allosteric agonist of CXCR7, and inhibits replication of HIV-1 and HIV-2 (EC_{50}=1-10nM). Plerixafor blocks CXCL12 binding to CXCR4, disrupts the anchoring of hematopoietic stem/progenitor cells to the bone marrow stromal microenvironment, thereby promoting CD34+ cell release into peripheral blood, and further affects tumor cell migration, immune cell infiltration and inflammatory responses. Plerixafor can be used in research related to hematopoietic stem cell mobilization for non-Hodgkin lymphoma and multiple myeloma, HIV infection, leukemia and other hematologic malignancies, solid tumors, as well as inflammation and immune modulation[1-4].

In vitro, treatment with Plerixafor (1nM-10μM) for 72h in Ewing sarcoma cell lines (A673, TC-32, TC-71, CADO-ES1, VH-64 cells) caused a dose-dependent increase in cell number and induced cell migration[5]. Treatment with Plerixafor (10μM) for 24h in human prostate cancer tumor endothelial cells (NEC and TEC cells) inhibited proliferation of tumor endothelial cells and reduced their migration ability[6]. Treatment with 1μg/ml Plerixafor for 24h in human colon cancer cells (LoVo)

significantly suppressed cell viability, proliferation, migration and invasion[7].

In vivo, BALB/c mice receiving intra-tibial injection of KLF5KQ cells were intraperitoneally administered Plerixafor at 3.5mg/kg daily for 28 or 32 days suppressed bone metastatic tumor growth and reduced osteoclast occurrence at the tumor-bone interface[8]. CD46 transgenic mice received a single subcutaneous injection of Plerixafor at 5mg/kg followed 45 minutes later by a subcutaneous injection of MGTA-145 at 2.5mg/kg, achieving efficient hematopoietic stem cell mobilization with approximately 25000 LSK cells per mL in peripheral blood[9]. C3H/HeJ mice received a single subcutaneous injection of Plerixafor at 5mg/kg, and rapid mobilization of CFU-GM, BFU-E and CFU-GEMM hematopoietic progenitor cells into peripheral blood occurred within 1 hour[10].

References:

[1] De Clercq E. Recent advances on the use of the CXCR4 antagonist plerixafor (AMD3100, Mozobil) and potential of other CXCR4 antagonists as stem cell mobilizers. Pharmacol Ther. 2010;128(3):509-518.

[2] Wang J, Tannous BA, Poznansky MC, et al. CXCR4 antagonist AMD3100 (plerixafor): From an impurity to a therapeutic agent. Pharmacol Res. 2020;161:105127.

[3] Jantunen E, Turunen A, Partanen A. Impact of plerixafor use in the mobilization of blood grafts for autologous hematopoietic cell transplantation. Transfusion. 2024;64(4):742-750.

[4] Steinberg M, Silva M. Plerixafor: A chemokine receptor-4 antagonist for mobilization of hematopoietic stem cells for transplantation after high-dose chemotherapy for non-Hodgkin's lymphoma or multiple myeloma. Pharmacotherapy. 2010;30(6):606-615.

[5] Berning P, Schaefer C, Clemens D, et al. The CXCR4 antagonist plerixafor (AMD3100) promotes proliferation of Ewing sarcoma cell lines in vitro and activates receptor tyrosine kinase signaling. Cell Commun Signal. 2018;16:21.

[6] Heidegger I, Fotakis G, Offermann A, et al. Comprehensive characterization of the prostate tumor microenvironment identifies CXCR4/CXCL12 crosstalk as a novel antiangiogenic therapeutic target in prostate cancer. Mol Cancer. 2022;21:132.

[7] Ma G, Jiang J, He T, et al. G-CSF promotes H3K27ac-modified KLF5 to activate CXCR4

expression and drive colon cancer growth and metastasis. Eur J Med Res. 2026;31(1):207.

[8] Zhang B, Li Y, Wu Q, et al. Acetylation of KLF5 maintains EMT and tumorigenicity to cause chemoresistant bone metastasis in prostate cancer. Nat Commun. 2021 Mar 17;12:1714.

[9] Li C, Goncalves KA, Rask T, et al. Single-dose MGTA-145/plerixafor leads to efficient mobilization and in vivo transduction of HSCs with thalassemia correction in mice. Blood Adv. 2021 Mar 9;5(5):1239-1249.

[10] Broxmeyer HE, Orschell CM, Clapp DW, et al. Rapid mobilization of murine and human hematopoietic stem and progenitor cells with AMD3100, a CXCR4 antagonist. J Exp Med. 2005 Apr 18;201(8):1307-1318.