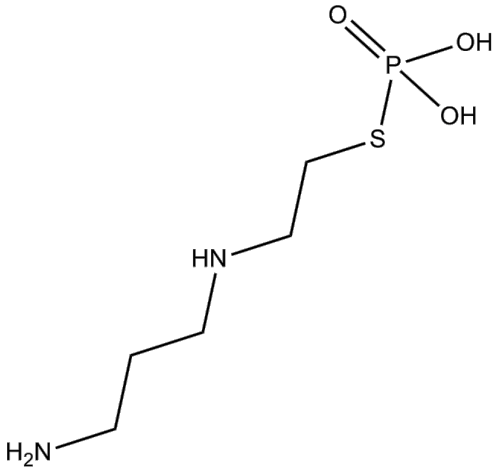
Product Data Sheet

Product Name: Amifostine
Cat. No.: GC15473

Chemical Properties

Cas. No.	20537-88-6		
Chemical Name	S-(2-((3-aminopropyl)amino)ethyl) O,O-dihydrogen phosphorothioate		
SMILES	NCCCNCCSP(O)(O)=O		
Formula	$C_5H_{15}N_2O_3PS$	M.Wt	214.22
Solubility	≥ 8.05mg/mL in Water	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 Dami cells

Product Data Sheet

Preparation Method	The Dami cells were cultured in RPMI 1640 medium containing 10% heat-inactivated fetal bovine serum (FBS) and 100U/ml of double antibiotics, under conditions of 37°C, 5% CO_2 and 100% humidity for cultivation. Dami cells were inoculated into 6-well plates with a density of 5×10^5 cells/ml. The cells were subcultured every 2 days. The concentration of Amifostine in the culture medium was 0.01, 0.1, 1, and 5mM. The cell count and cell morphology were analyzed every two days, and the entire experiment lasted for 12 days.
Reaction Conditions	0.01, 0.1, 1, and 5mM; 12 days
Applications	Amifostine treatment at a concentration higher than 1mM significantly inhibited the proliferation of Dami cells, caused changes in cell morphology.

Animal experiment [2]:

Animal models	Male C57BL/6J mice
Preparation Method	Adult male, 8-10-week old C57BL/6J mice, with an average weight of 20-25g, were anaesthetised with an intraperitoneal injection of ketamine (75mg/kg) and acepromazine (1.5mg/kg). LPS (0.7mg/kg) or sterile water was injected intratracheally in a small volume (20-30ml) using a 20-gauge catheter. Although water is a hypotonic solvent, it did not induce any noticeable injury in control animals injected with 20ml sterile water, when compared with untreated mice or mice injected with 20ml of normotonic physiological solution (data not shown). Mice were randomized to concurrently receive sterile saline solution or Amifostine (200mg/kg) by intraperitoneal injection to yield the experimental groups: (1) control; (2) LPS (0.7mg/kg) only; (3) Amifostine (200mg/kg) only; and (4) LPS (0.7mg/kg)+Amifostine (200mg/kg). After 18 hours, the lung tissues of the mice were collected for analysis.

Caution: Product has not been fully validated for medical applications. For research use only.
Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

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Dosage form	200mg/kg for once; 18h; i.p.
Applications	Amifostine treatment attenuated LPS-induced lung vascular leak and inhibited neutrophil accumulation in the lung parenchyma within mice.

References:
[1] Wang H, Yang B, Hu B, et al. The effect of amifostine on differentiation of the human megakaryoblastic Dami cell line[J]. Cancer Medicine, 2016, 5(8): 2012-2021.
[2] Fu P, Birukova A A, Xing J, et al. Amifostine reduces lung vascular permeability via suppression of inflammatory signalling[J]. European Respiratory Journal, 2009, 33(3): 612-624.

Background

Amifostine, an inorganic thiophosphate, is a selective broad-spectrum cytoprotector of normal tissues[1]. Amifostine is dephosphorylated by alkaline phosphatase in the body to form the active metabolite WR-1065, which scavenges oxygen free radicals and enhances the DNA repair process [2]. Amifostine has been widely used to reduce the severity of acute oral mucositis caused by radiation[3].

In vitro, Amifostine treatment for 5 days significantly inhibited the proliferation of myelodysplastic syndrome (MDS) cells, with an IC_{50} value of 14μM[4]. Treatment with 100μg/ml Amifostine for 12 hours inhibited apoptosis of 32D cells and enhanced the nuclear level of NF-κB/Rel[5]. Amifostine treatment for 12 days at a concentration of 1mM significantly inhibited the proliferation of Dami cells, caused changes in cell morphology[6].

In vivo, Amifostine treatment at a single dose of 200mg/kg for 18h attenuated lipopolysaccharide (LPS)-induced lung vascular leak and inhibited neutrophil accumulation in the lung parenchyma within mice[7]. A single subcutaneous injection of Amifostine (200mg/kg) for 30 minutes later significantly induced the expression of HIF1α in the tissues of normal rats[8].

References:
[1] Koukourakis M I. Amifostine in clinical oncology: current use and future applications[J]. Anti-

cancer drugs, 2002, 13(3): 181-209.
[2] Van den Berg J H, Beijnen J H, Balm A J M, et al. Future opportunities in preventing cisplatin induced ototoxicity[J]. Cancer treatment reviews, 2006, 32(5): 390-397.
[3] Kalman N S, Zhao S S, Anscher M S, et al. Current status of targeted radioprotection and radiation injury mitigation and treatment agents: a critical review of the literature[J]. International Journal of Radiation Oncology* Biology* Physics, 2017, 98(3): 662-682.
[4] Ribizzi I, Darnowski J W, Goulette F A, et al. Amifostine cytotoxicity and induction of apoptosis in a human myelodysplastic cell line[J]. Leukemia research, 2000, 24(6): 519-525.
[5] Romano M F, Lamberti A, Bisogni R, et al. Amifostine inhibits hematopoietic progenitor cell apoptosis by activating NF-κB/Rel transcription factors[J]. Blood, The Journal of the American Society of Hematology, 1999, 94(12): 4060-4066.
[6] Wang H, Yang B, Hu B, et al. The effect of amifostine on differentiation of the human megakaryoblastic Dami cell line[J]. Cancer Medicine, 2016, 5(8): 2012-2021.
[7] Fu P, Birukova A A, Xing J, et al. Amifostine reduces lung vascular permeability via suppression of inflammatory signalling[J]. European Respiratory Journal, 2009, 33(3): 612-624.
[8] Koukourakis M I, Giatromanolaki A, Chong W, et al. Amifostine induces anaerobic metabolism and hypoxia-inducible factor 1α[J]. Cancer chemotherapy and pharmacology, 2004, 53(1): 8-14.