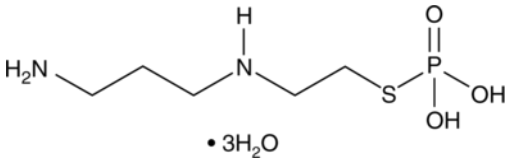
Product Data Sheet

Product Name: Amifostine (hydrate)
Cat. No.: GC42785

Chemical Properties

Cas. No.	112901-68-5		
SMILES	NCCCNCCSP(O)(O)=O.O.O.O		
Formula	C5H15N2O3PS•3H2O	M.Wt	268.3
Solubility	PBS (pH 7.2): 5 mg/ml	Storage	Store at 2-8°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	H9c2 cardiomyocytes (rat cardiac cell line)
Preparation Method	H9c2 cells were maintained in high-glucose Dulbecco's Modified Eagle Medium (DMEM) supplemented with 10% fetal bovine serum (FBS) at 37°C, 5% CO_2. Cells were pretreated with Amifostine (25–100μM) for 30 minutes before exposure to tert-butyl hydroperoxide (TBHP; 100μM) for 12 hours to simulate oxidative stress.

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Reaction Conditions	25–100μM; 30min pretreatment
Applications	Amifostine pretreatment significantly attenuated TBHP-induced cell death and apoptosis, accompanied by reduced reactive oxygen species (ROS) production and preservation of mitochondrial membrane potential (ΔΨm).

Animal experiment [2]:

Animal models	C57BL/6 mice
Preparation Method	Mice were intratracheally instilled with Bleomycin (3U/kg) to induce pulmonary fibrosis. Amifostine (200mg/kg) was administered intraperitoneally on days 1, 3, and 5 post-bleomycin challenge. Lung tissues and bronchoalveolar lavage fluid (BALF) were collected on days 7 and 21 for analysis.
Dosage form	200mg/kg; i.p.; Three doses.
Applications	Amifostine significantly attenuated bleomycin-induced pulmonary fibrosis, reduced lung inflammation (e.g., decreased inflammatory cells and cytokines in BALF), and improved survival rates. Amifostine also suppressed fibrosis markers (collagen, fibronectin, α-SMA) in lung tissues.

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References:
[1] Wu SZ, Tao LY, Wang JN, et al. Amifostine Pretreatment Attenuates Myocardial Ischemia/Reperfusion Injury by Inhibiting Apoptosis and Oxidative Stress. Oxid Med Cell Longev. 2017;2017:4130824.
[2] Guo F, Xu F, Li S, et al. Amifostine ameliorates bleomycin-induced murine pulmonary fibrosis via NAD+/SIRT1/AMPK pathway-mediated effects on mitochondrial function and cellular metabolism. Eur J Med Res. 2024 Jan 20;29(1):68.

Background

Amifostine (hydrate) is a broad-spectrum cytoprotective agent primarily used to reduce the toxicity of chemotherapy or radiotherapy on normal tissues[1-2]. Amifostine is a prodrug that is metabolized in vivo by alkaline phosphatase into the active thiol metabolite WR-1065. WR-1065 scavenges free radicals generated by cisplatin or radiation, promotes DNA repair, thereby reducing the toxicity of chemotherapeutic agents on the kidneys, bone marrow, and nerves, and alleviating xerostomia induced by radiotherapy[3-4].

In vitro, pretreatment of murine myeloid progenitor 32D cells with Amifostine (100μg/mL) for 18 hours, followed by culture under growth factor deprivation conditions, Amifostine significantly inhibited cell apoptosis and attenuated caspase-3 activity. Amifostine promoted the nuclear translocation of NF-κB/Rel by reducing cytoplasmic IκBα levels[5]. In H9c2 cardiomyocytes, pretreatment with Amifostine (25-100μM) for 30 minutes, followed by incubation with Tert-butyl hydroperoxide (TBHP; 100μM) for 12 hours, Amifostine significantly reduced cell apoptosis and oxidative stress[6].

In vivo, intraperitoneal injection of Amifostine (200mg/kg) in bleomycin (3U/kg)-induced pulmonary fibrosis C57BL6/J mice (administered on days 1, 3, and 5 post-modeling), Amifostine significantly alleviated pulmonary inflammation and fibrosis, improved mitochondrial function, and restored cellular metabolic balance via the NAD^+/SIRT1/AMPK pathway[7]. Subcutaneous injection of Amifostine (25mg/kg) in Oxaliplatin-induced peripheral sensory neuropathy Swiss albino mice (administered 30 minutes before each Oxaliplatin injection, twice a week for nine times), Amifostine significantly prevented mechanical hyperalgesia and cold allodynia, and reduced the expression of c-Fos,

nitrotyrosine, and ATF3 in the dorsal root ganglia[8].

References:

[1] Capizzi RL, Scheffler BJ, Schein PS. Amifostine-mediated protection of normal bone marrow from cytotoxic chemotherapy. Cancer. 1993 Dec 1;72(11 Suppl):3495-501.
[2] Koukourakis MI. Amifostine in clinical oncology: current use and future applications. Anticancer Drugs. 2002 Mar;13(3):181-209.
[3] Kouvaris JR, Kouloulias VE, Vlahos LJ. Amifostine: the first selective-target and broad-spectrum radioprotector. Oncologist. 2007 Jun;12(6):738-47.
[4] Singh VK, Seed TM. The efficacy and safety of amifostine for the acute radiation syndrome. Expert Opin Drug Saf. 2019 Nov;18(11):1077-1090.
[5] Romano MF, Lamberti A, Bisogni R, et al. Amifostine inhibits hematopoietic progenitor cell apoptosis by activating NF-kappaB/Rel transcription factors. Blood. 1999 Dec 15;94(12):4060-6.
[6] Wu SZ, Tao LY, Wang JN, et al. Amifostine Pretreatment Attenuates Myocardial Ischemia/Reperfusion Injury by Inhibiting Apoptosis and Oxidative Stress. Oxid Med Cell Longev. 2017;2017:4130824.
[7] Guo F, Xu F, Li S, et al. Amifostine ameliorates bleomycin-induced murine pulmonary fibrosis via NAD+/SIRT1/AMPK pathway-mediated effects on mitochondrial function and cellular metabolism. Eur J Med Res. 2024 Jan 20;29(1):68.
[8] Pereira AF, Lino JA, Alves BWF, et al. Amifostine protects from the peripheral sensory neuropathy induced by oxaliplatin in mice. Braz J Med Biol Res. 2020 Sep 18;53(11):e10263.

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