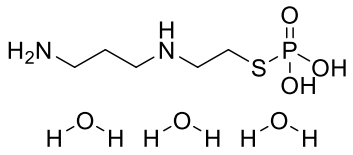
Product data sheet

MedKoo Cat#: 100050 Name: Amifostine trihydrate CAS#: 112901-68-5 (trihydrate) Chemical Formula: $C_5H_{21}N_2O_6PS$ Molecular Weight: 268.26	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:

Amifostine is a phosphorylated aminosulfhydryl compound. After dephosphorylation of amifostine by alkaline phosphatase to an active free sulfhydryl (thiol) metabolite, the thiol metabolite binds to and detoxifies cytotoxic platinum-containing metabolites of cisplatin and scavenges free radicals induced by cisplatin and ionizing radiation. The elevated activity of this agent in normal tissues results from both the relative abundance of alkaline phosphatase in normal tissues and the greater vascularity of normal tissues compared to tumor tissues.

2. CoA, QC data, SDS, and handling instruction

SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
PBS (pH 7.2)	5.0	18.64
Water	42.0	156.56

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	3.73 mL	18.64 mL	37.28 mL
5 mM	0.75 mL	3.73 mL	7.46 mL
10 mM	0.37 mL	1.86 mL	3.73 mL
50 mM	0.07 mL	0.37 mL	0.75 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator

Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

1. Huang B, He T, Yao Q, Zhang L, Yao Y, Tang H, Gong P. Amifostine Suppresses the Side Effects of Radiation on BMSCs by Promoting Cell Proliferation and Reducing ROS Production. Stem Cells Int. 2019 Jan 9;2019:8749090. doi: 10.1155/2019/8749090. PMID: 30728842; PMCID: PMC6343176.
2. Wang HT, Yang B, Hu B, Chi XH, Luo LL, Yang HQ, Lang XL, Geng J, Qiao CX, Li Y, Wu XX, Zhu HL, Lv M, Lu XC. The effect of amifostine on differentiation of the human megakaryoblastic Dami cell line. Cancer Med. 2016 Aug;5(8):2012-21. doi: 10.1002/cam4.759. Epub 2016 May 26. PMID: 27228575; PMCID: PMC4884634.

In vivo study

1. Cheng H, Lv M, Mi R, Xue G. Amifostine ameliorates cerebral ischaemia-reperfusion injury via p38-mediated oxidative stress and mitochondrial dysfunction. Folia Neuropathol. 2020;58(4):334-346. doi: 10.5114/fn.2020.102436. PMID: 33480238.
2. Pereira AF, Lino JA, Alves BWF, Lisboa MRP, Pontes RB, Leite CAVG, Nogueira RB, Lima-Júnior RCP, Vale ML. Amifostine protects from the peripheral sensory neuropathy induced by oxaliplatin in mice. Braz J Med Biol Res. 2020 Sep 18;53(11):e10263. doi: 10.1590/1414-431X202010263. PMID: 32965323; PMCID: PMC7510240.

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7. Bioactivity

Biological target:

Amifostine trihydrate is a hypoxia-inducible factor-α1 (HIF-α1) and p53 inducer.

In vitro activity

As depicted in Figure 2(a), there was no difference in the proliferation of cells treated with 0 M, 10^{-7} M, and 10^{-9} M AMI (Amifostine). However, the proliferation of cells in the group treated with 10^{-5} M AMI was significantly inhibited on the seventh day of the experiment. Therefore, AMI was administered at a concentration of 10^{-7} M in the subsequent experiments. Figure 2(b) demonstrates that the proliferation of BMSCs in group C was significantly inhibited in comparison to that of group A ($P < 0.05$) on the fifth and seventh days of the experiment. The proliferation of cells on the fifth and seventh days was much higher in the group that received 2 Gy radiation in conjunction with AMI than the group that received 2 Gy radiation alone ($P < 0.05$).

Reference: Stem Cells Int. 2019; 2019: 8749090. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6343176/

In vivo activity

Strikingly, amifostine treatment markedly reduced neurological deficits and appeared to be dose-dependent (Fig. 1A). The percentage of brain infarct area was obviously enlarged in the MCAO/R group, while amifostine treatment apparently diminished it, particularly in the MCAO/R + H group (Fig. 1C). Also, there was a significant increase in brain water content of MCAO/R mice, which could be relieved by amifostine treatment (Fig. 1D). Furthermore, H&E staining displayed the histopathological and morphological alteration of hippocampus CA1 (Fig. 1E). In the sham group, the neuron cell structure was normal, the edge was clear, and the nuclei and intercellular substance were stained evenly. In contrast, the neurons in the MCAO/R group exhibited a disordered arrangement with obvious oedema and necrosis. Importantly, treatment of amifostine significantly attenuated the extent of brain oedema and neuronal necrosis. By using Nissl staining, the results showed that the number of stained Nissl's bodies was significantly declined in mice of the MCAO/R group. After amifostine treatment, its number was increased significantly (Fig. 1F). These findings provided evidence that amifostine could prevent brain failure caused by I/R.

Reference: Folia Neuropathol. 2020;58(4):334-346. https://pubmed.ncbi.nlm.nih.gov/33480238/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.