Amifostine thiol dihydrochloride

Cat. No.: HY-103640

CAS No.: 14653-77-1

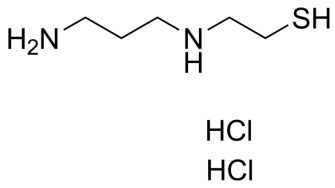
Molecular Formula: $C_5H_{16}Cl_2N_2S$

Molecular Weight: 207.16

Target: MDM-2/p53

Pathway: Apoptosis

Storage: -20°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 125 mg/mL (603.40 mM; Need ultrasonic)

Preparing Stock Solutions	Concentration / Solvent / Mass	1 mg	5 mg	10 mg
	1 mM	4.8272 mL	24.1359 mL	48.2719 mL
	5 mM	0.9654 mL	4.8272 mL	9.6544 mL
	10 mM	0.4827 mL	2.4136 mL	4.8272 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1.67 mg/mL (8.06 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.67 mg/mL (8.06 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1.67 mg/mL (8.06 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Amifostine thiol (WR-1065) dihydrochloride can protect normal tissues from the toxic effects of certain cancer agents and activate p53 through a JNK-dependent signaling pathway.

IC_{50} & Target

p53[1]

In Vitro

The DNA-binding activity is increased in a Amifostine thiol dihydrochloride (Amifostine thiol) concentration-dependent manner. Cells treated with 1 mM Amifostine thiol dihydrochloride for 24 h reveal that all of the p53-induced genes analyzed are transactivated following Amifostine thiol dihydrochloride treatment, in a p53-dependent manner. Significantly, treatment with Amifostine thiol dihydrochloride leads to a 3-fold increase in luciferase expression driven by AP-1, and a 5-

	fold increase when this reporter gene is driven by NF-κB, when these values are normalized to the level of the cotransfected β-galactosidase gene[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	The results show that Amifostine thiol dihydrochloride (Amifostine thiol) attenuates the severity of 6-OHDA-induced catalepsy (P<0.001) when compare with 6-OHDA-lesioned rats. Also it has been observed that Amifostine thiol dihydrochloride improves catalepsy in dose dependent manner (P<0.001). Pretreatment with three different doses of Amifostine thiol dihydrochloride (20, 40 and 80 μg/2 μL/rat) for 3 days before 6-OHDA administration, significantly (P<0.001) elevates SOD activity and restores it to normal range compare with 6-OHDA lesioned rats[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [2]	For Western analysis, cells are treated with 1 mM WR-1065 dihydrochloride (WR-1065) for 24 h, and subconfluent cultures of cells are harvested and lysed in RIPA buffer supplemented with protease inhibitors. Protein concentrations are determined by a detergent-compatible assay. Western blots are blocked and incubated in antibody in PBS/0.2% Tween 20/5% nonfat dry milk. Blots are incubated with 1 μg/mL antibody for 1 h at room temperature, followed by washing in PBS/0.2% Tween 20 and incubation in peroxidase-conjugated secondary antibody and chemiluminescence detection[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [2]	To test the effects of paclitaxel in the presence or absence of WR-1065 dihydrochloride (WR-1065) on cell growth, cells are seeded in 96-well tissue culture dishes at 20% confluence and allowed to attach and recover for at least 24 h. Varying combinations of paclitaxel alone or in combination with a 60 min pretreatment with 1 mM WR-1065 dihydrochloride are then added to each well, and the plates are incubated for an additional 48 h or 72 h. The number of surviving cells is determined by staining. The percentage of cells killed by paclitaxel and/or WR-1065 dihydrochloride is calculated as the percentage decrease in sulforhodamine B binding compare with control cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Seventy two rats are divided randomly into 9 equal groups: 1) Control group receives no injection and is left untreated for the entire period of the experiment as intact animals; 2) Sham operated group is subjected only to surgical procedure; 3) Vehicle (saline)-treated group receives 2 μL saline (intra-SNc); 4) Lesioned group receives 6-hydroxydopamine; 5) Vehicle+6OHDA group receives saline as a vehicle 3 days once daily (2 μL/rat) before 6-OHDA injection; 6 to 8) Rats in these groups are pretreated with intra-SNc injection of WR-1065 dihydrochloride (WR-1065) (20, 40 and 80 μg/2 μL/rat) 3 days before 6-OHDA injection; 9) Non-lesioned animals receive intra-SNc injection of WR-1065 dihydrochloride (80 μg/2 μL/rat) for three days[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Front Cell Dev Biol. 2020 Jul 29;8:703.

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REFERENCES

[1]. Pluquet O, et al. The cytoprotective aminothiol WR1065 activates p53 through a non-genotoxic signaling pathway involving c-Jun N-terminal kinase. J Biol Chem. 2003 Apr 4;278(14):11879-87.

[2]. Shen H, et al. Binding of the aminothiol WR-1065 to transcription factors influences cellular response to anticancer drugs. J Pharmacol Exp Ther. 2001 Jun;297(3):1067-

73.

[3]. Afshin Kheradmand, et al. Effect of WR-1065 on 6-hydroxydopamine-induced catalepsy and IL-6 level in rats. Iran J Basic Med Sci. 2016 May; 19(5): 490-496.

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