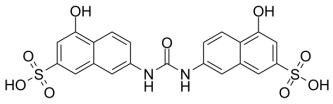
AMI-1 free acid

Cat. No.:	HY-18962A
CAS No.:	134-47-4
Molecular Formula:	$C_{21}H_{16}N_2O_9S_2$
Molecular Weight:	504.49
Target:	Histone Methyltransferase
Pathway:	Epigenetics
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 83.33 mg/mL (165.18 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9822 mL	9.9110 mL	19.8220 mL
5 mM	0.3964 mL	1.9822 mL	3.9644 mL
10 mM	0.1982 mL	0.9911 mL	1.9822 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: ≥ 2.08 mg/mL (4.12 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.12 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

AMI-1 free acid is a potent, cell-permeable and reversible inhibitor of protein arginine N-methyltransferases (PRMTs), with IC_{50}s of 8.8 μM and 3.0 μM for human PRMT1 and yeast-Hmt1p, respectively. AMI-1 free acid exerts PRMTs inhibitory effects by blocking peptide-substrate binding[1].

IC_{50} & Target

IC50: 8.8 μM (PRMT1), 3.0 μM (yeast-Hmt1p)[1]

In Vitro

AMI-1 free acid can inhibit the in vitro methylation reactions performed by all five recombinantly active PRMTs (PRMT1, -3, -4, and -6 and Hmt1p)[2].
AMI-1 free acid not only inhibits type I PRMTs (PRMT1, 3, 4 and 6) but also type II PRMT5[2].
AMI-1 free acid specifically inhibits arginine, but not lysine, methyltransferase activity in vitro and does not compete for the

AdoMet binding site[3].
AMI-1 free acid inhibits methylation of GFP-Npl3 and cellular proteins[3].
AMI-1 free acid (0.6-2.4 mM; 48-96 hours) inhibits the cell viability of sarcoma in S180 and U2OS cells in a time-dependent and dose-dependent manner in vitro[4].
AMI-1 free acid (1.2-2.4 mM; 48-72 hours) reduces S180 cell viability through the induction of cell apoptosis[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[4]

Cell Line:	S180 cells, U2OS cells
Concentration:	0.6 mM, 1.2 mM, 2.4 mM
Incubation Time:	48 hours, 72 hours, 96 hours
Result:	Inhibited the cell viability.

Apoptosis Analysis[4]

Cell Line:	S180 cells
Concentration:	1.2 mM, 2.4 mM
Incubation Time:	48 hours, 72 hours
Result:	Increased the percentages of cells undergoing apoptosis.

In Vivo

AMI-1 free acid (0.5 mg; intratumorally; daily; for 7 days) inhibits S180 viability in vivo[4].
AMI-1 free acid (0.5 mg; intratumorally; daily; for 7 days) downregulates PRMT5 but does not regulate the expression of PRMT7 in a tumor xenograft model[4].
AMI-1 free acid (0.5 mg; intratumorally; daily; for 7 days) decreases the levels of H4R3me2s and H3R8me2s in a tumor xenograft model[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	6-7 weeks old male Kunming mice (18-22 g), with S180 cells xenograft[4]
Dosage:	0.5 mg
Administration:	Intratumorally, daily, for 7 days
Result:	Decreased tumor weight.

CUSTOMER VALIDATION

- Nat Commun. 2023 Feb 23;14(1):1011.
- Cell Death Dis. 2023 Sep 22;14(9):624.
- Genes Dis. 2023 Mar 28.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Donghang Cheng, et al. Small Molecule Regulators of Protein Arginine Methyltransferases. J Biol Chem. 2004 Jun 4;279(23):23892-9.

[2]. Zhang, B., et al. Targeting protein arginine methyltransferase 5 inhibits colorectal cancer growth by decreasing arginine methylation of eIF4E and FGFR3. Oncotarget. 2015 Sep 8;6(26):22799-811.

[3]. Baolai Zhang, et al. Arginine Methyltransferase inhibitor-1 Inhibits Sarcoma Viability in vitro and in vivo. Oncol Lett. 2018 Aug;16(2):2161-2166.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA