Amsacrine

Cat. No.: HY-13551

CAS No.: 51264-14-3

Molecular Formula: $C_{21}H_{19}N_3O_3S$

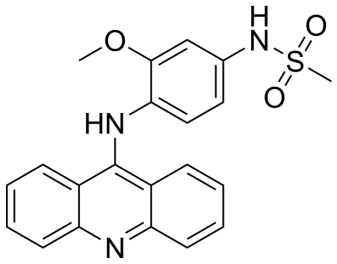
Molecular Weight: 393.46

Target: Topoisomerase; Autophagy

Pathway: Cell Cycle/DNA Damage; Autophagy

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 : 9.3 mg/mL (23.64 mM; Need ultrasonic and warming)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5416 mL	12.7078 mL	25.4155 mL
	5 mM	0.5083 mL	2.5416 mL	5.0831 mL
	10 mM	0.2542 mL	1.2708 mL	2.5416 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: ≥ 2.5 mg/mL (6.35 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Amsacrine (m-AMSA; acridinyl anisidide) is an inhibitor of topoisomerase II, and acts as an antineoplastic agent which can intercalates into the DNA of tumor cells.

IC_{50} & Target

Topoisomerase II

In Vitro

Amsacrine (m-AMSA) blocks HERG currents in HEK 293 cells and Xenopus oocytes in a concentration-dependent manner, with IC_{50} values of 209.4 nm and 2.0 μM, respectively. Amsacrine (m-AMSA) causes a negative shift in the voltage dependence of both activation (−7.6 mV) and inactivation (−7.6 mV). HERG current block by amsacrine is not frequency dependent[1]. In vitro studies of normal human lymphocytes with various concentrations of Amsacrine (m-AMSA), show both increased levels of chromosomal aberrations, ranging from 8% to 100%, and increase SCEs, ranging from 1.5 times the normal at the lowest concentration studied (0.005 μg/mL) to 12 times the normal (0.25 μg/mL)[3]. Amsacrine (m-AMSA)-induced apoptosis of U937 cells is characterized by caspase-9 and caspase-3 activation, increased intracellular Ca^{2+}

	concentration, mitochondrial depolarization, and MCL1 down-regulation. Amsacrine (m-AMSA) induces MCL1 down-regulation by decreasing its stability. Further, amsacrine-treated U937 cells show AKT degradation and Ca^{2+}-mediated ERK inactivation[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	In animals treated with different doses of amsacrine (0.5-12 mg/kg), the frequencies of micronucleated polychromatic erythrocytes increase significantly after treatment with 9 and 12 mg/kg. Furthermore, the present study demonstrates for the first time that Amsacrine (m-AMSA) has high incidences of clastogenicity and low incidences of aneugenicity whereas nocodazole has high incidences of aneugenicity and low incidences of clastogenicity during mitotic phases in vivo[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [2]	Amsacrine (m-AMSA) is investigated in three separated experiments. In the first experiment, animals are treated by intraperitoneal injection with 0.5, 1.5 and 4.5 mg/kg of amsacrine and bone marrow is sampled 24 h after treatment. Preliminary negative MN results at this sampling time lead to the use of 30 h sampling time for amsacrine. Thus, in the second experiment, mice are treated with 0.5, 1.5 and 4.5 mg/kg of Amsacrine (m-AMSA) and bone marrow is sampled 30 h after treatment. The doses and sampling times for amsacrine are chosen by reference to earlier studies and the selected doses are within the dose range used for human chemotherapy. The results again show that the micronuclei frequency in the bone marrow of mice is not affected by treatment with any of the selected doses of the test agent, at 30 h sampling time, thus, in the third experiment, mice are treated with 6, 9 and 12 mg/kg of amsacrine and bone marrow is sampled 24 and 30 h after treatment. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Metab. 2022 Feb 7;34(3):424-440.e7.
- J Adv Res. 2023 Jun 6;S2090-1232(23)00148-0.
- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.
- Nucleic Acid Ther. 2023 Jun 30.
- bioRxiv. September 29, 2021.

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REFERENCES

[1]. Thomas D, et al. Inhibition of cardiac HERG currents by the DNA topoisomerase II inhibitor amsacrine: mode of action. Br J Pharmacol. 2004 Jun;142(3):485-94.

[2]. Attia SM. Molecular cytogenetic evaluation of the mechanism of genotoxic potential of amsacrine and nocodazole in mouse bone marrow cells. J Appl Toxicol. 2013 Jun;33(6):426-33.

[3]. Kao-Shan CS, et al. Cytogenetic effects of amsacrine on human lymphocytes in vivo and in vitro. Cancer Treat Rep. 1984 Jul-Aug;68(7-8):989-97.

[4]. Lee YC, et al. Amsacrine-induced apoptosis of human leukemia U937 cells is mediated by the inhibition of AKT- and ERK-induced stabilization of MCL1. Apoptosis. 2016 Oct 19

Caution: Product has not been fully validated for medical applications. For research use only.

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