Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

Amsacrine (hydrochloride)

Catalog No: tcsc1943

Available Sizes

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
54301-15-4

Formula:
$C_{21}H_{20}ClN_3O_3S$

Pathway:
Cell Cycle/DNA Damage;Autophagy

Target:
Topoisomerase;Autophagy

Purity / Grade:
>98%

Solubility:
10 mM in DMSO

Alternative Names:
m-AMSA hydrochloride; acridinyl anisidide hydrochloride

Observed Molecular Weight:
429.92

Product Description

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Amsacrine (mAMSA) hydrochloride is an inhibitor of **topoisomerase II**, and acts as an antineoplastic agent which can intercalates into the DNA of tumor cells.

IC50 & Target: Topoisomerase II[1]

In Vitro: Amsacrine (mAMSA) 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 (mAMSA) causes a negative shift in the voltage dependence of both activation (−7.6 mV) and inactivation (−7.6 mV). HERG current block by Amsacrine (mAMSA) is not frequency dependent[1]. In vitro studies of normal human lymphocytes with various concentrations of Amsacrine (mAMSA), 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 (mAMSA)-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 induces MCL1 down-regulation by decreasing its stability. Further, amsacrine-treated U937 cells show AKT degradation and Ca^{2+}-mediated ERK inactivation[4].

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 (mAMSA) 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].