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

Amsacrine

Catalog No: tcsc1942

Available Sizes

Size: 10mg

Size: 50mg

Size: 100mg

Size: 500mg

Specifications

CAS No:
51264-14-3

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

Pathway:
Cell Cycle/DNA Damage;Autophagy

Target:
Topoisomerase;Autophagy

Purity / Grade:
>98%

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

Alternative Names:
m-AMSA;acridinyl anisidide

Observed Molecular Weight:
393.46

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

Product Description

Amsacrine (m-AMSA) 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 (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].

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].