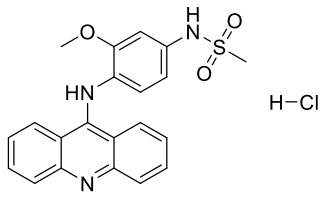
Product data sheet

MedKoo Cat#: 100057 Name: Amsacrine HCl CAS#: 54301-15-4 (HCl) Chemical Formula: $C_{21}H_{20}ClN_3O_3S$ Molecular Weight: 429.92	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:
Amsacrine is an aminoacridine derivative with potential antineoplastic activity. Although its mechanism of action is incompletely defined, amsacrine may intercalate into DNA and inhibit topoisomerase II, resulting in DNA double-strand breaks, arrest of the S/G2 phase of the cell cycle, and cell death. This agent's cytotoxicity is maximal during the S phase of the cell cycle when topoisomerase levels are greatest. In addition, amsacrine may induce transcription of tumor promoter p53 protein and block p53 ubiquitination and proteasomal degradation, resulting in p53-dependent tumor cell apoptosis.

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	37.25	86.64
DMSO:PBS (pH 7.2) (1:1)	0.5	1.16
DMF	10.0	23.26

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.33 mL	11.63 mL	23.26 mL
5 mM	0.47 mL	2.33 mL	4.65 mL
10 mM	0.23 mL	1.16 mL	2.33 mL
50 mM	0.05 mL	0.23 mL	0.47 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Liu WH, Chen YJ, Chien JH, Chang LS. Amsacrine suppresses matrix metalloproteinase-2 (MMP-2)/MMP-9 expression in human leukemia cells. J Cell Physiol. 2014 May;229(5):588-98. doi: 10.1002/jcp.24481. PMID: 24122234.

In vivo study

1. Yamamoto K, Kokubun T, Sato K, Akaishi T, Shimazaki A, Nakamura M, Shiga Y, Tsuda S, Omodaka K, Saya H, Nakazawa T. The DNA topoisomerase II inhibitor amsacrine as a novel candidate adjuvant in a model of glaucoma filtration surgery. Sci Rep. 2019 Dec 17;9(1):19288. doi: 10.1038/s41598-019-55365-7. PMID: 31848363; PMCID: PMC6917768.
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. doi: 10.1002/jat.1753. Epub 2011 Nov 11. PMID: 22081495.

7. Bioactivity
Biological target:

Product data sheet

Amsacrine hydrochloride (m-AMSA hydrochloride; acridinyl anisidide hydrochloride) is an inhibitor of topoisomerase II.

In vitro activity

As shown in Figure 1B, cell treatment with m-AMSA dose ranging from 5 to 50 nM showed an approximately 6–12% decrease in U937, Jurkat, HL-60, KU812, and MEG-01 cell viability, while there was no significant loss in the viability of K562 cells after m-AMSA treatment. These results reflected that m-AMSA exerted marginal cytotoxicity on leukemia cells. Figure 1C shows that m-AMSA notably inhibited leukemia cell invasion. Western blot analysis showed that m-AMSA treatment reduced MMP-2 and MMP-9 protein expression (Fig. 1E). Figure 1F reveals that MMP-2 mRNA and MMP-9 mRNA levels in m-AMSA-treated cells were lower than those in untreated control cells as evidenced by real-time PCR assay. mRNA stability analysis following transcription inhibition by actinomycin D showed that m-AMSA notably reduced MMP-2/MMP-9 mRNA stability in U937, K562, and Jurkat cells as evidenced by real-time PCR analyses (Fig. 1H). These results indicated that m-AMSA-induced MMP-2/MMP-9 down-regulation inhibited leukemia cell invasion, and suggested that the m-AMSA suppressive effect on MMP-2/MMP-9 expression was associated with reduced MMP-2/MMP-9 transcription and mRNA stability.

Reference: J Cell Physiol. 2014 May;229(5):588-98. https://onlinelibrary-wiley-com.libproxy.lib.unc.edu/doi/10.1002/

In vivo activity

These results show that postoperative IOP (intraocular pressure) in rabbit eyes treated with 10% AMSA (amsacrine) was lower than in eyes treated with saline on day 7 after surgery. This suggests that AMSA is effective and begins to act during an early phase of wound healing. Furthermore, the difference in postoperative IOP between the 0.04% MMC and 10% AMSA groups started to widen after 21 days. This suggests that AMSA might also have an additional mechanism of action during the remodeling phase, which in rabbits corresponds to the period later than day 21 after surgery.

Reference: Sci Rep. 2019; 9: 19288. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6917768/