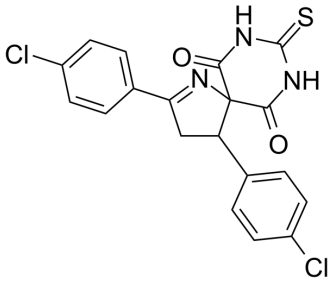
SCR130

Cat. No.:	HY-139297
CAS No.:	2377858-38-1
Molecular Formula:	$C_{19}H_{13}Cl_2N_3O_2S$
Molecular Weight:	418.3
Target:	DNA/RNA Synthesis; Apoptosis
Pathway:	Cell Cycle/DNA Damage; Apoptosis
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (239.06 mM; Need ultrasonic and warming)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.3906 mL	11.9531 mL	23.9063 mL
5 mM	0.4781 mL	2.3906 mL	4.7813 mL
10 mM	0.2391 mL	1.1953 mL	2.3906 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.98 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

SCR130 is a SCR7-based DNA nonhomologous end-joining (NHEJ) inhibitor. SCR130 inhibits the end-joining of DNA in a Ligase IV-dependent manner. SCR130 is specific to Ligase IV, and shows minimal or no effect on Ligase III and Ligase I mediated joining. SCR130 induces cell apoptosis and has anticancer activity[1].

In Vitro

SCR130 (7-21 μM; 48 hours) increase in the number of late and early apoptotic cells. SCR130 induces apoptosis by both intrinsic and extrinsic pathways. SCR130 increases the expression of p-p53, BCL2 and MCL1, and CYTOCHROME C, BAX, and BAK also increaseed. The activation of caspase 8, increase in expression of FAS and SMAC-DIABLO proteins are also observed [1].

SCR130 (48 hours) exhibits cytotoxicity in Reh, HeLa, CEM, Nalm6, and N114 cells with IC_{50} values of 14.1 μM, 5.9 μM, 6.5 μM, 2.2 μM, and 11 μM, respectively[1].

SCR130 can potentiate the effect of radiation (0.5 and 1 Gy) by inducing enhanced cell death upon coadministration in Reh and Nalm6 cell lines[1].

SCR130 blocks the endogenous NHEJ leading to accumulation of unrepaired DNA breaks. Treatment with SCR130 leads to inhibition of endogenous NHEJ, resulting in the accumulation of DNA double-strand breaks (DSBs) and cell death by activating apoptotic pathways[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Apoptosis Analysis[1]

Cell Line:	Reh cells
Concentration:	7 μM, 14 μM, and 21 μM
Incubation Time:	48 hours
Result:	Showed a concentration-dependent increase in the number of late and early apoptotic cells.

Western Blot Analysis[1]

Cell Line:	Reh cells
Concentration:	7 μM, 14 μM, and 21 μM
Incubation Time:	48 hours
Result:	Revealed a concentration-dependent increase in levels of pATM and activation of p53 through phosphorylation.

REFERENCES

[1]. Ujjayinee Ray, et al. Identification and characterization of novel SCR7-based small-molecule inhibitor of DNA end-joining, SCR130 and its relevance in cancer therapeutics. Mol Carcinog. 2020 Jun;59(6):618-628.

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