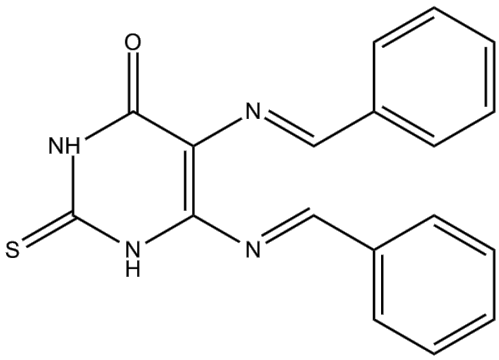
Product Data Sheet

Product Name: SCR7
Cat. No.: GC12106

Chemical Properties

Cas. No.	1533426-72-0		
Chemical Name	5,6-bis((E)-benzylideneamino)-2-thioxo-2,3-dihydropyrimidin-4(1H)-one		
SMILES	S=C(NC(/N=C/C1=CC=CC=C1)=C2/N=C/C3=CC=CC=C3)NC2=O		
Formula	$C_{18}H_{14}N_4OS$	M.Wt	334.39
Solubility	≥ 16.7195mg/mL in DMSO	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

Cell experiment [1]:

Cell lines A549

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Preparation Method	A549 cells were treated with an increasing concentration of SCR7 (10, 50, 100, and 250μM) for 48h. Cell proliferation was then evaluated by MTT assay. The medium was then changed with MTT solution (0.5mg/ml) in DMEM or RPMI without phenol red and FBS; cells were incubated at 37°C for 30min and violet precipitate was dissolved with isopropanol. Absorbance at 550nm was measured through a Plate reader.
Reaction Conditions	10, 50, 100, and 250μM, 48h
Applications	SCR7 can induced cytotoxicity in A549 cells.

Animal experiment [1]:

Animal models	BALB/c mice bearing EAC tumors
Preparation Method	Mice bearing EAC tumors were treated with SCR7 (10mg/kg，six doses). Ki67 (a cell proliferation marker), p21 (cell cycle regulator) and apoptotic markers such as BID and Caspase 3 were measured in treated sections by immunohistochemistry.
Dosage form	10mg/kg, every alternate day for six doses, i.p.
Applications	Accumulation of DSBs upon SCR7 treatment activates p53-mediated intrinsic pathway of apoptosis in BALB/c mice.

Caution: Product has not been fully validated for medical applications. For research use only.
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References:
[1]. Srivastava M, Nambiar M, Sharma S, et al. An inhibitor of nonhomologous end-joining abrogates double-strand break repair and impedes cancer progression. Cell. 2012 Dec 21;151(7):1474-87. doi: 10.1016/j.cell.2012.11.054.

Background

SCR7 is a small molecule inhibitor of DNA Ligase IV [1] [2], which inhibits the non-homologous end-joining (NHEJ) pathway by binding to the DNA binding domain (DBD) of Ligase IV [3]. SCR7 is commonly used in cancer therapeutic research [4] and serves as a valuable tool for improving the efficiency of genome editing [5].

SCR7 exhibits concentration- and time-dependent cytotoxicity in DLBCL cells (SUDHL8) via both extrinsic and intrinsic apoptosis pathways. In SUDHL8 cells, treatment with SCR7 at 100 and 250μM for 48 and 72 hours increases mitochondrial membrane potential, causes translocation of phosphatidylserine to the cell surface, and raises the expression of double-strand break repair proteins and apoptotic markers [6].

SCR7 (10mg/kg, every alternate day for six doses, i.p.) treatment can significantly enhance the the effect of radiation in mice with Dalton's lymphoma (DLA) liquid tumors. The combination of SCR7 and ionizing radiation (IR) can markedly reduce tumor growth and improve the survival rate of tumor-bearing mice [7].

References:
[1]. Srivastava M, Nambiar M, Sharma S, et al. An inhibitor of nonhomologous end-joining abrogates double-strand break repair and impedes cancer progression. Cell. 2012 Dec 21;151(7):1474-87. doi: 10.1016/j.cell.2012.11.054.
[2]. Gkotzamanidou M, Terpos E, Bamia C, et al. DNA repair of myeloma plasma cells correlates with clinical outcome: the effect of the nonhomologous end-joining inhibitor SCR7. Blood. 2016 Sep 1;128(9):1214-25. doi: 10.1182/blood-2016-01-691618. Epub 2016 Jul 21. PMID: 27443291; PMCID: PMC5524533.
[3]. Manjunath M, Choudhary B, Raghavan SC. SCR7, a potent cancer therapeutic agent and a biochemical inhibitor of nonhomologous DNA end-joining. Cancer Rep (Hoboken). 2021

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[4]. Vartak SV, Swarup HA, Gopalakrishnan V, et al. Autocyclized and oxidized forms of SCR7 induce cancer cell death by inhibiting nonhomologous DNA end joining in a Ligase IV dependent manner. FEBS J. 2018 Nov;285(21):3959-3976. doi: 10.1111/febs.14661. Epub 2018 Oct 8. PMID: 30230716.
[5]. Hu Z, Shi Z, Guo X, Jiang B, Wang G, Luo D, Chen Y, Zhu YS. Ligase IV inhibitor SCR7 enhances gene editing directed by CRISPR-Cas9 and ssODN in human cancer cells. Cell Biosci. 2018 Feb 19;8:12. doi: 10.1186/s13578-018-0200-z. PMID: 29468011; PMCID: PMC5819182.
[6]. Gopalakrishnan V, Radha G, Raghavan SC, et al. Inhibitor of nonhomologous end joining can inhibit proliferation of diffuse large B-Cell lymphoma cells and potentiate the effect of ionization radiation. J Radiat Cancer Res 2018;9:93-101. DOI: 10.4103/jrcr.jrcr_9_18
[7]. Gopalakrishnan V, Sharma S, Ray U, et al. SCR7, an inhibitor of NHEJ can sensitize tumor cells to ionization radiation. Mol Carcinog. 2021 Sep;60(9):627-643. doi: 10.1002/mc.23329. Epub 2021 Jun 30.

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