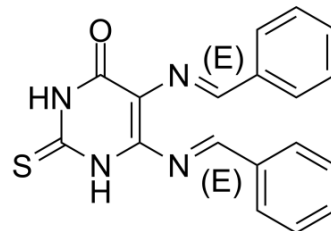


## SCR7

<b>Cat. No.:</b>	HY-12742		
<b>CAS No.:</b>	1533426-72-0		
<b>Molecular Formula:</b>	C <sub>18</sub> H <sub>14</sub> N <sub>4</sub> OS		
<b>Molecular Weight:</b>	334.39		
<b>Target:</b>	DNA/RNA Synthesis; CRISPR/Cas9; Apoptosis		
<b>Pathway:</b>	Cell Cycle/DNA Damage; Apoptosis		
<b>Storage:</b>	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



## SOLVENT & SOLUBILITY

### In Vitro

DMSO : ≥ 45 mg/mL (134.57 mM)  
 \* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent		1 mg	5 mg	10 mg
	Concentration	Mass			
	1 mM		2.9905 mL	14.9526 mL	29.9052 mL
	5 mM		0.5981 mL	2.9905 mL	5.9810 mL
	10 mM		0.2991 mL	1.4953 mL	2.9905 mL

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

### In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline  
 Solubility: ≥ 2.5 mg/mL (7.48 mM); Clear solution

## BIOLOGICAL ACTIVITY

### Description

SCR7 is an unstable form that can be autocyclized into a stable form SCR7 pyrazine. SCR7 pyrazine is a DNA ligase IV inhibitor that blocks nonhomologous end-joining (NHEJ) in a ligase IV-dependent manner. SCR7 pyrazine is also a CRISPR/Cas9 enhancer which increases the efficiency of Cas9-mediated homology-directed repair (HDR). SCR7 pyrazine induces cell apoptosis and has anticancer activity<sup>[1][2]</sup>.

### IC<sub>50</sub> & Target

DNA Ligase IV      CRISPR/Cas9

### In Vitro

SCR7 (SCR7 pyrazine; 20-100 μM; 24 hours; MCF7 cells) treatment interferes with NHEJ in cells, leading to accumulation of unrepaired double-strand breaks (DSBs)<sup>[1]</sup>.

SCR7 (SCR7 pyrazine) treatment shows a dose-dependent decrease in cell proliferation with IC<sub>50</sub> values of 40 μM, 34 μM, 44 μM, 8.5 μM, 120 μM, 10 μM and 50 μM for MCF7, A549, HeLa, T47D, A2780, HT1080 and Nalm6 cells, respectively<sup>[1]</sup>.

In MCF7 cells, SCR7 (SCR7 pyrazine; 20, 40  $\mu$ M) treatment increases phosphorylation of ATM and activates p53, decreases MDM2, BCL2, resulting in activation of proapoptotic proteins, PUMA and BAX. And the shorter fragments of MCL1, PARP1, Caspase 3, and Caspase 9 cleavage are upregulated in a dose-dependent manner<sup>[1]</sup>.

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

#### Western Blot Analysis<sup>[1]</sup>

Cell Line:	MCF7 cells
Concentration:	20 $\mu$ M, 40 $\mu$ M, 100 $\mu$ M
Incubation Time:	24 hours
Result:	Showed an increase in levels of gH2AX foci and protein.

#### In Vivo

SCR7 (SCR7 pyrazine; 10 mg/kg; intraperitoneal injection; six doses; BALB/c mice) treatment significantly reduces breast adenocarcinoma-induced tumor and increases lifespan<sup>[1]</sup>.

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

Animal Model:	BALB/c mice injected with breast adenocarcinoma cells <sup>[1]</sup>
Dosage:	10 mg/kg
Administration:	Intraperitoneal injection; on alternate days (0, 2, 4, 6, 8, and 10)
Result:	Significantly reduced breast adenocarcinoma-induced tumor and increased lifespan.

## CUSTOMER VALIDATION

- EMBO Rep. 2019 Mar;20(3):e46821.
- J Genet Genomics. 2021 Mar 30.
- J Immunol. 2020 Apr 1;204(7):1904-1918.
- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.
- Onco Targets Ther. 2018 Aug 17;11:4945-4953.

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## REFERENCES

- [1]. Srivastava M, 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.
- [2]. Lin C, et al. Increasing the Efficiency of CRISPR/Cas9-mediated Precise Genome Editing of HSV-1 Virus in Human Cells. Sci Rep. 2016 Oct 7;6:34531.
- [3]. Supriya V Vartak, 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.

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**Caution: Product has not been fully validated for medical applications. For research use only.**

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