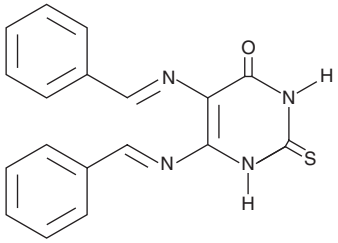
PRODUCT INFORMATION

SCR7

Item No. 18015

CAS Registry No.:	1533426-72-0
Formal Name:	2,3-dihydro-5,6-*bis*[(E)-(phenylmethylene)amino]-2-thioxo-4(1H)-pyrimidinone
MF:	$C_{18}H_{14}N_4OS$
FW:	334.4
Purity:	≥95%
UV/Vis.:	λ_{max}: 312, 387 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

SCR7 is supplied as a crystalline solid. A stock solution may be made by dissolving the SCR7 in the solvent of choice, which should be purged with an inert gas. SCR7 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of SCR7 in these solvents is approximately 15, 10, and 20 mg/ml, respectively.

SCR7 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, SCR7 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. SCR7 has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

DNA ligase IV seals double-strand breaks during the process of nonhomologous end-joining in DNA repair. Inhibiting this function in cancer cells is one strategy to prevent deleterious cell growth. SCR7 is a small molecule inhibitor of DNA ligase IV that prevents nonhomologous end-joining by interfering with ligase binding and activating apoptosis.[1] It also inhibits ligase III, but does not affect the activity of T4 DNA ligase or ligase I. SCR7 has been used to increase the rate of homology directed repair triggered by DNA double-strand breaks and to inhibit cancer cell growth *in vitro* (IC_{50}s = 8-120 µM) and in mouse models when co-administered with double-strand break-inducing therapeutic compounds.[1-4]

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* **151(7)**, 1474-1487 (2012).
2. Chu, V.T., Weber, T., Wefers, B., *et al*. Increasing the efficiency of homology-directed repair for CRISPR-Cas9-induced precise gene editing in mammalian cells. *Nat. Biotech.* **33(5)**, 543-550 (2015).
3. John, F., George, J., Srivastava, M., *et al*. Pluronic copolymer encapsulated SCR7 as a potential anticancer agent. *Faraday Discussions* **177**, 1-7 (2014).
4. Maruyama, T., Dougan, S.K., Truttmann, M.C., *et al*. Increasing the efficiency of precise genome editing with CRISPR-Cas9 by inhibition of nonhomologous end joining. *Nat. Biotech.* **33(5)**, 538-542 (2015).

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