Small Molecules

SCR7

NHEJ pathway inhibitor; inhibits DNA ligase IV

Catalog # 74102 5 mg
74104 10 mg

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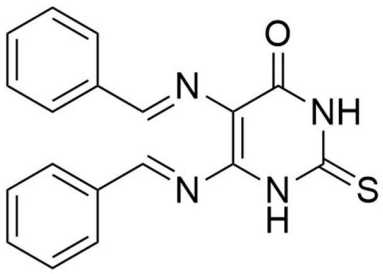
Product Description

SCR7 is an inhibitor of DNA ligase IV, which is responsible for the repair of DNA double-strand breaks via the non-homologous end joining (NHEJ) repair pathway (Srivastava et al.). Due to its reported success in impeding cancer cell growth and potential impact on future cancer therapeutics, SCR7 has been closely studied in many recent publications (Hosoya & Miyagawa; John et al.).

Molecular Name: SCR7
Alternative Names: Not applicable
CAS Number: 1533426-72-0
Chemical Formula: $C_{18}H_{14}N_4OS$
Molecular Weight: 334.4 g/mol
Purity: ≥ 95%
Chemical Name: 5-(benzylideneamino)-6-[(E)-benzylideneamino]-2-sulfanylidene-1H-pyrimidin-4-one
Structure:



Properties

Physical Appearance: A crystalline solid

Storage: Product stable at -20°C as supplied. Protect product from prolonged exposure to light. For long-term storage, store with a desiccant.
Stable as supplied for 12 months from date of receipt.

Solubility:
· DMSO ≤ 25 mM
· Absolute ethanol ≤ 40 mM
· DMF ≤ 55 mM
For example, to prepare a 10 mM stock solution in DMSO, resuspend 1 mg in 299 µL of DMSO.

Prepare stock solution fresh before use. Information regarding stability of small molecules in solution has rarely been reported, however, as a general guide we recommend storage in DMSO at -20°C. Aliquot into working volumes to avoid repeated freeze-thaw cycles. The effect of storage of stock solution on compound performance should be tested for each application.

Compound has low solubility in aqueous media. For use as a cell culture supplement, stock solution should be diluted into culture medium immediately before use. Avoid final DMSO concentration above 0.1% due to potential cell toxicity.

Published Applications

GENOME EDITING
·Inhibits NHEJ-dependent DNA repair; this inhibition is reported to enhance precise homology-directed repair (HDR)-dependent CRISPR-Cas9 genome editing (Chu et al.; Maruyama et al.; Pinder et al.). However, these effects are cell type-specific and context-dependent (Song et al.; Xie et al.; Yang et al.; Zhang et al.).
CANCER RESEARCH
·Activates apoptosis of cancer cells by inhibiting DNA ligase IV to increase the efficacy of DNA double-strand break-inducing therapy (chemo- or radio-therapy) (Srivastava et al.).

References

Chu VT et al. (2015) Increasing the efficiency of homology-directed repair for CRISPR-Cas9-induced precise gene editing in mammalian cells. Nat Biotechnol 33(5): 543–8.
Hosoya N & Miyagawa K. (2014) Targeting DNA damage response in cancer therapy. Cancer Sci 105(4): 370–88.
John F et al. (2015) Enhanced efficacy of pluronic copolymer micelle encapsulated SCR7 against cancer cell proliferation. Macromol Biosci 15(4): 521–34.
Maruyama T et al. (2015) Increasing the efficiency of precise genome editing with CRISPR-Cas9 by inhibition of nonhomologous end joining. Nat Biotechnol 33(5): 538–42.
Pinder J et al. (2015) Nuclear domain “knock-in” screen for the evaluation and identification of small molecule enhancers of CRISPR-based genome editing. Nucleic Acids Res 43(19): 9379–92.
Song J et al. (2016) RS-1 enhances CRISPR/Cas9- and TALEN-mediated knock-in efficiency. Nat Commun 7: 10548.
Srivastava M et al. (2012) An inhibitor of nonhomologous end-joining abrogates double-strand break repair and impedes cancer progression. Cell 151(7): 1474–87.
Xie Z et al. (2017) Optimization of a CRISPR/Cas9-mediated knock-in strategy at the porcine Rosa26 locus in porcine foetal fibroblasts. Sci Rep 7(1): 3036.
Yang D et al. (2016) Enrichment of G2/M cell cycle phase in human pluripotent stem cells enhances HDR-mediated gene repair with customizable endonucleases. Sci Rep 6: 21264.
Zhang J-P et al. (2017) Efficient precise knockin with a double cut HDR donor after CRISPR/Cas9-mediated double-stranded DNA cleavage. Genome Biol 18(1): 35.

Related Small Molecules

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