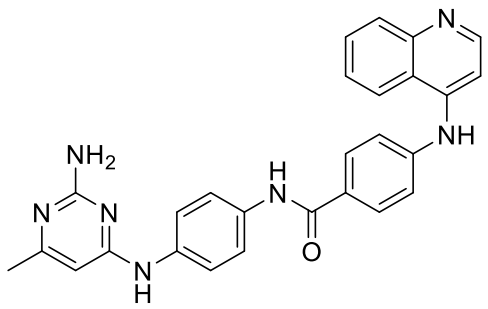
Product data sheet

MedKoo Cat#: 406558 Name: SGI-1027 CAS#: 1020149-73-8 Chemical Formula: $C_{27}H_{23}N_7O$ Exact Mass: 461.1964 Molecular Weight: 461.52	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:
SGI-1027 is a potent DNMT inhibitor, which reactivates tumor suppressor genes by blocking DNA methyltransferase 1 activity and inducing its degradation. Treatment of different cancer cell lines with SGI-1027 resulted in selective degradation of DNMT1 with minimal or no effects on DNMT3A and DNMT3B. At a concentration of 2.5 to 5 micromol/L (similar to that of decitabine), complete degradation of DNMT1 protein was achieved within 24 h without significantly affecting its mRNA level. SGI-1027 may have the potential for use in epigenetic cancer therapy.

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	20.0	43.30

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.17 mL	10.83 mL	21.67 mL
5 mM	0.43 mL	2.17 mL	4.33 mL
10 mM	0.22 mL	1.08 mL	2.17 mL
50 mM	0.04 mL	0.22 mL	0.43 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Li JJ, Ryou CS, Kim DH. [Effects of SGI-1027 on Formation and Elimination of PrP^(Sc) in Prion-Infected Cells]. Mol Biol (Mosk). 2020 May-Jun;54(3):469-473. Russian. doi: 10.31857/S0026898420030118. PMID: 32492010.
2. Sun N, Zhang J, Zhang C, Zhao B, Jiao A. DNMTs inhibitor SGI-1027 induces apoptosis in Huh7 human hepatocellular carcinoma cells. Oncol Lett. 2018 Nov;16(5):5799-5806. doi: 10.3892/ol.2018.9390. Epub 2018 Sep 4. PMID: 30344731; PMCID: PMC6176375.

In vivo study

1. Hong L, Chen W, He L, Tan H, Peng D, Zhao G, Shi X, Wang L, Liu M, Jiang H. Effect of Naoluoxintong on the NogoA/RhoA/ROCK pathway by down-regulating DNA methylation in MCAO rats. J Ethnopharmacol. 2021 Dec 5;281:114559. doi: 10.1016/j.jep.2021.114559. Epub 2021 Aug 28. PMID: 34461189.
2. Reyes-Aguirre LI, Lamas M. Oct4 Methylation-Mediated Silencing As an Epigenetic Barrier Preventing Müller Glia Dedifferentiation in a Murine Model of Retinal Injury. Front Neurosci. 2016 Nov 15;10:523. doi: 10.3389/fnins.2016.00523. PMID: 27895551; PMCID: PMC5108807.

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7. Bioactivity

Biological target:

SGI-1027 is a DNA methyltransferase inhibitor that inhibits the mammalian DNA methyltransferases DNMT3B, DNMT3A and DNMT1 (IC50s = 7.5, 8 and 12.5 μM, respectively, with Poly(dl-dC) as the substrate). SGI-1027 reactivates silenced tumor suppressor genes by reducing CpG island hypermethylation.

In vitro activity

SGI-1027 can be used in the management of human hepatocellular carcinoma (HCC). It causes cell apoptosis via the mitochondrial-mediated pathway. In the Huh7 cell line, SGI-1027 treatment resulted in a significant dose-dependent decrease in cell viability. SGI-1027 resulted in cell apoptosis and typical apoptotic nucleic alterations. SGI-1027 downregulated B cell lymphoma-2 expression and upregulated Bcl-associated X protein expression. However, no significant alterations of the cell cycle phases were observed.

Reference: Oncol Lett. 2018 Nov;16(5):5799-5806. https://pubmed.ncbi.nlm.nih.gov/30344731/

In vivo activity

Intravitreous administration of SGI-1027 induced Oct4 expression at 24 hours post-injury (hpi) in Müller glia (MG), indicating that DNA methylation may play a regulatory role in the response of MG to injury. Oct4 is rapidly expressed after retinal injury but is silenced at 24 hpi. This silencing correlates with a decrease in the expression of the Dnmt3b, returning to basal levels at 24 hpi. Changes in Oct4 methylation levels indicate that DNA methylation may restrict the injury-induced dedifferentiation of MG.

Reference: Front Neurosci. 2016 Nov 15;10:523. https://pubmed.ncbi.nlm.nih.gov/27895551/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.