Product data sheet



MedKoo Cat#: 406624			
Name: SC66			
CAS#: 871361-88-5			
Chemical Formula: C ₁₈ H ₁₆ N ₂ O			
Exact Mass: 276.1263			
Molecular Weight: 276.33			
Product supplied as:	Powder		
Purity (by HPLC):	≥ 98%	$\neg \dot{N} \cdot \neg \dot{N} \cdot \neg \dot{N} = \neg \dot{N} \cdot \neg $	
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:

SC66 is a novel and potent AKT inhibitor, which reduced cell viability in a dose- and time-dependent manner, inhibited colony formation and induced apoptosis in HCC cells. SC66 treatment led to a reduction in total and phospho-AKT levels. SC66 significantly potentiated the effects of both conventional chemotherapeutic and targeted agents, doxorubicin and everolimus, respectively. In vivo, SC66 inhibited tumor growth of Hep3B cells in xenograft models, with a similar mechanism observed in the in vitro model. SC66 had antitumor effects on HCC cells.

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	25	90.47

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	3.62 mL	18.09 mL	36.19 mL
5 mM	0.72 mL	3.62 mL	7.24 mL
10 mM	0.36 mL	1.81 mL	3.62 mL
50 mM	0.07 mL	0.36 mL	0.72 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. Wang L, Song R, Ma M, Chen Y, Jiang Y, Li J, Yang Z, Zhang L, Jing M, Wang X, Zhang M, Fan J. Inhibition of autophagy can promote the apoptosis of bladder cancer cells induced by SC66 through the endoplasmic reticulum stress pathway. Chem Biol Interact. 2023 Oct 1;384:110725. doi: 10.1016/j.cbi.2023.110725. Epub 2023 Sep 21. PMID: 37741534.
- Gao L, Liu J, Xu P, Deng G, Liu B, Yuan F, Tan Y, Sun Q, Xu Y, Zhang H, Qi Y, Han S, Yang K, Geng R, Jiang H, Chen Q. AKT Inhibitor SC66 Inhibits Proliferation and Induces Apoptosis in Human Glioblastoma Through Down-Regulating AKT/β-Catenin Pathway. Front Pharmacol. 2020 Jul 31;11:1102. doi: 10.3389/fphar.2020.01102. PMID: 32848734; PMCID: PMC7411127.

In vivo study

1. Liu Y, Huang Y, Ding J, Liu N, Peng S, Wang J, Wang F, Zhang Y. Targeting Akt by SC66 triggers GSK-3β mediated apoptosis in colon cancer therapy. Cancer Cell Int. 2019 May 9;19:124. doi: 10.1186/s12935-019-0837-7. PMID: 31168297; PMCID: PMC6509835.

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2. Wu YH, Huang YF, Chen CC, Chou CY. Akt inhibitor SC66 promotes cell sensitivity to cisplatin in chemoresistant ovarian cancer cells through inhibition of COL11A1 expression. Cell Death Dis. 2019 Apr 11;10(4):322. doi: 10.1038/s41419-019-1555-8. PMID: 30975980; PMCID: PMC6459878.

7. Bioactivity

Biological target:

SC66 reduces cell viability in a dose- and time-dependent manner, inhibits colony formation, and induces apoptosis in hepatocellular carcinoma cells.

In vitro activity

SC66 is a promising novel agent for patients with bladder cancer. SC66 inhibited cell proliferation, triggered mitochondria-mediated apoptosis, and initiated autophagy in bladder cancer cells. Study results suggested that SC66-caused apoptosis and autophagy were endoplasmic reticulum stress-dependent. Autophagy activation can partially protect bladder cancer cells from apoptosis under endoplasmic reticulum stress induced by SC66 treatment.

Reference: Chem Biol Interact. 2023 Oct 1;384:110725. https://pubmed.ncbi.nlm.nih.gov/37741534/

In vivo activity

SC66 has potential in therapeutic treatment for ovarian cancer. A NOD-SCID xenograft mouse model demonstrated that SC66 treatment caused a reduction in tumor formation and enhanced the therapeutic efficacy of cisplatin. SC66-sensitized chemoresistant cells to cisplatin and paclitaxel treatment, and promoted apoptosis.

Reference: Cell Death Dis. 2019 Apr 11;10(4):322. https://pubmed.ncbi.nlm.nih.gov/30975980/

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.