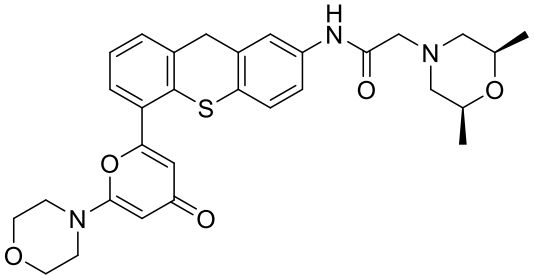


Product data sheet



MedKoo Cat#: 406441 Name: KU-60019 CAS: 925701-46-8 Chemical Formula: C ₃₀ H ₃₃ N ₃ O ₅ S Exact Mass: 547.2141 Molecular Weight: 547.67	
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:

KU-60019 is a potent and selective ATM inhibitor. KU-60019 is 10-fold more effective than KU-55933 at blocking radiation-induced phosphorylation of key ATM targets in human glioma cells. As expected, KU-60019 is a highly effective radiosensitizer of human glioma cells. KU-60019 inhibits the DNA damage response, reduces AKT phosphorylation and prosurvival signaling, inhibits migration and invasion, and effectively radiosensitizes human glioma 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
DMF	20.0	36.52
DMF:PBS (pH 7.2) (1:1)	0.5	0.91
DMSO	75.0	136.94
Ethanol	50.0	91.30

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	1.83 mL	9.13 mL	18.26 mL
5 mM	0.37 mL	1.83 mL	3.65 mL
10 mM	0.18 mL	0.91 mL	1.83 mL
50 mM	0.04 mL	0.18 mL	0.37 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. Golding SE, Rosenberg E, Adams BR, Wignarajah S, Beckta JM, O'Connor MJ, Valerie K. Dynamic inhibition of ATM kinase provides a strategy for glioblastoma multiforme radiosensitization and growth control. *Cell Cycle*. 2012 Mar 15;11(6):1167-73. doi: 10.4161/cc.11.6.19576. Epub 2012 Mar 15. PMID: 22370485; PMCID: PMC3335919.
2. Golding SE, Rosenberg E, Valerie N, Hussaini I, Frigerio M, Cockcroft XF, Chong WY, Hummersone M, Rigoreau L, Menear KA, O'Connor MJ, Povirk LF, van Meter T, Valerie K. Improved ATM kinase inhibitor KU-60019 radiosensitizes glioma cells, compromises insulin, AKT and ERK prosurvival signaling, and inhibits migration and invasion. *Mol Cancer Ther*. 2009 Oct;8(10):2894-902. doi: 10.1158/1535-7163.MCT-09-0519. Epub 2009 Oct 6. PMID: 19808981; PMCID: PMC2761990.

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In vivo study

1. McCabe N, Hanna C, Walker SM, Gonda D, Li J, Wikstrom K, Savage KI, Butterworth KT, Chen C, Harkin DP, Prise KM, Kennedy RD. Mechanistic Rationale to Target PTEN-Deficient Tumor Cells with Inhibitors of the DNA Damage Response Kinase ATM. *Cancer Res.* 2015 Jun 1;75(11):2159-65. doi: 10.1158/0008-5472.CAN-14-3502. Epub 2015 Apr 13. PMID: 25870146.

7. Bioactivity

Biological target:

KU-60019 is an improved ATM kinase-specific inhibitor with IC₅₀ of 6.3 nM.

In vitro activity

The second generation ATMi analog KU-60019 provided quick, reversible and complete inhibition of the DDR at sub-micromolar concentrations in human glioblastoma cells. KU-60019 inhibited the phosphorylation of the major DNA damage effectors p53, H2AX and KAP1 as well as AKT.

Reference: *Cell Cycle.* 2012 Mar 15;11(6):1167-73. <https://pubmed.ncbi.nlm.nih.gov/22370485/>

In vivo activity

Lastly, the ATM inhibitor KU-60019 was specifically toxic to PTEN mutant cancer cells in mouse tumor xenografts and reversible by reintroduction of wild-type PTEN.

Reference: *Cancer Res.* 2015 Jun 1;75(11):2159-65. <https://pubmed.ncbi.nlm.nih.gov/25870146/>

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.