Product Data Sheet

Product Name: KMR-206
Cat. No.: GC79056

Chemical Properties

Cas. No.	2992741-10-1		
Formula	$C_{29}H_{23}FN_6O_2$	M.Wt	506.53
Solubility	DMSO: 50 mg/mL (98.71 mM; Need ultrasonic)	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure

Background

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KMR-206 is a PARP7 inhibitor with an IC_{50} of 13.7 nM. KMR-206 relieves AHR -mediated transcriptional repression and enhances CYP1A1 expression in the presence of TCDD. KMR-206 induces the STING -dependent IFN-β signaling pathway and increases the levels of STAT1, pSTAT1 and nuclear PARP7 in cancer cells. KMR-206 reduces the viability of lung adenocarcinoma cells, enhances radiation-induced immunogenic signals, and induces the production of immunogenic signals in glioblastoma cancer stem cells. KMR-206 destabilizes FRA1 to increase IRF1 levels and promotes the IRF3 - CBP / p300 interaction. KMR-206 can be used in studies related to lung adenocarcinoma and glioblastoma [1] [2].

In Vitro, KMR-206 potently and selectively inhibits the catalytic activity of purified PARP7 in vitro, with an IC_{50} of 13.7 nM[1]. KMR-206 (0.001-0.3 μM; duration sufficient to measure MARylation) inhibits the auto-MARylation of GFP-PARP7 in HEK 293T cells with an EC50 of 8 nM, and upregulates GFP-PARP7 protein levels in a dose-dependent manner[1]. KMR-206 (0.1-1000 nM; 24 h) reverses AHR ligand-mediated transcriptional inhibition of CYP1A1 in WT MEFs in a dose-dependent manner in vitro, with a saturating effect observed at 100 nM[1]. KMR-206 (100 nM; 24 h) increases the IFN-β mRNA level in WT MEFs[1]. KMR-206 (0.01-0.3 μM; 16 h) dose-dependently induces the IFN-β signaling pathway in CT-26 cells, including upregulating STAT1/pSTAT1 levels, STING-dependent IFN-β secretion, and ISRE reporter gene activity[1]. KMR-206 (0.01-0.3 μM; 16 h) upregulates the endogenous PARP7 protein level in CT-26 cells in a dose-dependent manner[1]. KMR-206 (6 days) reduces the viability of NCI-H1373 cells in a dose-dependent manner with an EC50 of 104 nM, but exerts no effect on the viability of CT-26 cells[1]. KMR-206 (300 nM; imaged at 0, 30, 120 min post-treatment) alters the nuclear localization of GFP-PARP7 in HeLa cells, converting punctate nuclear localization into a diffuse pan-nuclear pattern within 30 min post-treatment[1]. KMR-206 (300 nM; added 2 h prior to irradiation and removed 24 h after irradiation) sensitizes human glioblastoma cell lines U251, T98G and KALS-1 to X-ray irradiation[2]. KMR-206 (administered 2 h before the first radiation fraction and maintained for 72 h after the last fraction) enhances radiotherapy-induced IFN-I and pro-inflammatory gene expression in U251, T98G and KALS-1 human glioblastoma cell lines, and exerts cell line-specific effects on gene targets and pathway enrichment[2]. KMR-206 (300 nM; added 2 h prior to irradiation and maintained for 72 h after irradiation) enhances radiation-induced activation of the IFN-I pathway in T98G cells and also enhances IFN-I pathway activation in KALS-1 cells[2]. Supernatants generated from the combined treatment of human glioblastoma cell lines

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U251, T98G and KALS-1 with KMR-206 (300 nM; added 2 h prior to the first radiation fraction and maintained for 72 h after the last fraction) and 3 × 8 Gy X-rays activate primary human NK, NKT and monocyte populations, with cell line-specific activation characteristics[2]. Combination of KMR-206 (administered 2 h before the first radiotherapy fraction and maintained for 72 h after the last fraction) with X-ray irradiation induces activation of the IFN-I pathway and expression of proinflammatory cytokines in STING-low-expressing LK17 patient-derived human glioblastoma cancer stem cells[2].

References:

[1]. Sanderson DJ, et al. Structurally distinct PARP7 inhibitors provide new insights into the function of PARP7 in regulating nucleic acid-sensing and IFN-β signaling. Cell Chem Biol. 2023;30(1):43-54.e8.
[2]. Lippert L, et al. PARP7 inhibition and a STING agonist potentiate radiation-induced immunogenicity in glioblastoma. Oncoimmunology. 2026;15(1):2656053.

Caution: Product has not been fully validated for medical applications. For research use only.
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