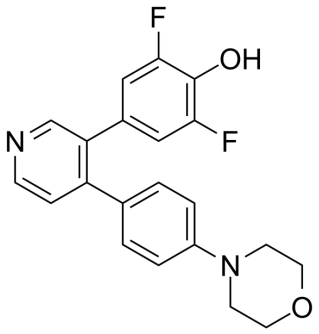
LJI308

Cat. No.:	HY-19713
CAS No.:	1627709-94-7
Molecular Formula:	$C_{21}H_{18}F_2N_2O_2$
Molecular Weight:	368.38
Target:	Ribosomal S6 Kinase (RSK); YB-1
Pathway:	MAPK/ERK Pathway; Cell Cycle/DNA Damage
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (67.86 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.7146 mL	13.5729 mL	27.1459 mL
	5 mM	0.5429 mL	2.7146 mL	5.4292 mL
	10 mM	0.2715 mL	1.3573 mL	2.7146 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.79 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

LJI308 is a potent pan-ribosomal S6 kinase (RSK) inhibitor, with IC_{50}s of 6 nM, 4 nM, and 13 nM for RSK1, RSK2, and RSK3, respectively. LJI308 inhibits the phosphorylation of RSK (T359/S363) and YB-1 (S102) after irradiation, treatment with EGF, and in cells expressing a KRAS mutation[1][2].

IC_{50} & Target	RSK1	RSK2	RSK3

In Vitro

LJI308 inhibits S6K1 with an IC_{50} of 0.8 μM[1].
LJI308 inhibits YB-1 phosphorylation in CRC cells at concentrations of 5 to 25 μM. In a dose kinetics experiment, LJI308, starting at 2.5 μM, inhibits YB-1 phosphorylation in the KRAS mutated TNBC cell line MDA-MB-231 by approximately 86%. LJI308 effectively blocks RSK and YB-1 phosphorylation after EGF stimulation and after irradiation in KRAS wild-type HBL-100 cells[2].
LJI308 (1-10 μM; 96 hours) decreases cell viability by up to 90%[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[3]

Cell Line:	HTRY-LT cell lines
Concentration:	1-10 μM
Incubation Time:	96 hours
Result:	Decreased cell viability by up to 90%.

CUSTOMER VALIDATION

- Circulation. 2022 Nov 30.
- Exp Hematol Oncol. 2023 Nov 30;12(1):100.
- J Invest Dermatol. 2020 Sep 9;S0022-202X(20)32055-8.
- Oncotarget. 2017 Jun 6;8(23):37633-37645.

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REFERENCES

[1]. Aronchik I, et al. Novel potent and selective inhibitors of p90 ribosomal S6 kinase reveal the heterogeneity of RSK function in MAPK-driven cancers. Mol Cancer Res. 2014 May;12(5):803-12.

[2]. Lettau K, et al. Simultaneous Targeting of RSK and AKT Efficiently Inhibits YB-1-Mediated Repair of Ionizing Radiation-Induced DNA Double-Strand Breaks in Breast Cancer Cells. Int J Radiat Oncol Biol Phys. 2021;109(2):567-580.

[3]. Jain R, et al. Discovery of Potent and Selective RSK Inhibitors as Biological Probes. J Med Chem. 2015 Sep 10;58(17):6766-83.

[4]. Davies AH, et al. Inhibition of RSK with the novel small-molecule inhibitor LJI308 overcomes chemoresistance by eliminating cancer stem cells. Oncotarget. 2015;6(24):20570-20577.

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