

Product Data Sheet

Product Name: KRIBB11

Cat. No.: GC19215

Chemical Properties

Cas. No. 342639-96-7

SMILES CNC1=CC=C([N+])([O-])=O)C(NC2=CC3=C(NN=C3)C=C2)=N1

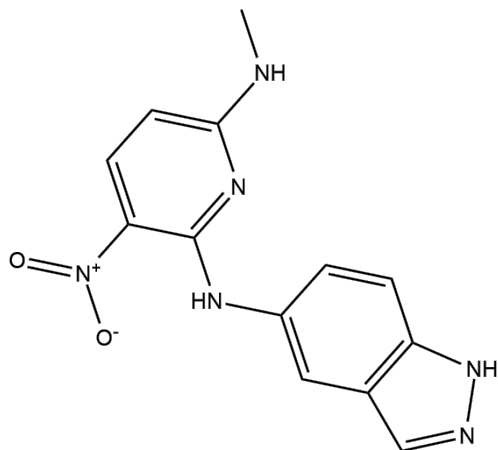
Formula $C_{13}H_{12}N_6O_2$ M.Wt 284.27

Solubility DMSO : ≥ 27 mg/mL (94.98 mM) 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



Protocol

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

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Product Data Sheet

Cell experiment [1]:	
Cell lines	A549 cells
Preparation Method	A549 cells were cultured in RPMI 1640 medium, supplemented 1% penicillin-streptomycin and 10% fetal bovine serum (FBS), in a humidified 5% CO ₂ incubator at 37°C. Cells were seeded at a density of 5×10 ³ cells per well into 96-well plates 24h. A549 cells were exposed to varying concentrations of KRIBB11 (0, 0.01, 0.05, 0.1, 0.5, 1, 5, 10, 25, and 50µM, respectively). After 24h of treatment, cell viability was calculated.
Reaction Conditions	0, 0.01, 0.05, 0.1, 0.5, 1, 5, 10, 25, and 50µM; 24h
Applications	KRIBB11 treatment reduced cell viability of A549 cells in a dose-dependent manner.
Animal experiment [2]:	
Animal models	LSL-Kras ^{G12D/+} ; Pdx1-Cre mice
Preparation Method	LSL-Kras ^{G12D/+} ; Pdx1-Cre mice (8 weeks of age) were fed ad libitum under controlled light-dark cycles (12h of light followed by 12h of dark) at 22±3°C. Mice were treated with KRIBB11 (50mg/kg/day; i.p.) for 1 month. After euthanasia, mouse pancreatic tissues were harvested for analysis.
Dosage form	50mg/kg/day; 1 month; i.p.

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Applications	KRIBB11 treatment suppressed tumorigenesis of pancreatic cancer in LSL-Kras ^{G12D/+} ; Pdx1-Cre mice.
References:[1] Zhang X, Lei Y, Chen X, et al. Suppression of NSCLC progression via the co-administration of Danusertib, an AURK inhibitor, and KRIBB11, an HSF1 inhibitor[J]. Biochemical Pharmacology, 2024, 223: 116155.[2] Qian W, Chen K, Qin T, et al. The EGFR-HSF1 axis accelerates the tumorigenesis of pancreatic cancer[J]. Journal of Experimental & Clinical Cancer Research, 2021, 40(1): 25.	

Background

KRIBB11 is a potent heat shock factor 1 (HSF1) inhibitor, with an IC₅₀ value of 1.2μM^[1]. KRIBB11 promotes the proteasomal degradation of Mcl-1 by mediating the ubiquitination increase through the E3 ubiquitin ligase Mule, thereby exerting an apoptotic effect^[2]. KRIBB11 has been widely used to promote microglial process elongation and suppress neuroinflammation^[3].

In vitro, KRIBB11 treatment for 48 hours significantly inhibited the viability of MCF-7, BT-474, and BT-20 cells, with IC₅₀ values of 3μM, 25μM, and 16μM, respectively^[4]. Treatment with 12.5μM KRIBB11 for 24h induced DNA damage, upregulated p53 and p21, and reduced the expression of cyclin D2/E, CDK2/4, c-Myc, MDM2, and β-catenin in human T-cell leukemia virus type 1 (HTLV-1)-infected T cells^[5]. 5μM of KRIBB11 incubation for 48 hours significantly promoted apoptosis of A172 cells, accompanied by accumulation of p53 and decrease in p27 levels^[6].

In vivo, a single intraperitoneal injection of KRIBB11 (5mg/kg) 1 hour before lipopolysaccharide (LPS) injection suppressed the expression of iNOS protein and mRNA in the brain tissue of endotoxemic mice^[7]. Intraperitoneal injection of KRIBB11 at a dose of 50mg/kg/day for 8 consecutive days, combined with 4 sessions of Modulated electro-hyperthermia (mEHT), inhibited tumor growth in the 4T1 xenograft mouse model^[8]. Continuous intraperitoneal injection of KRIBB11 (50mg/kg/day) for 30 consecutive days delayed the occurrence of pancreatic cancer in LSL-Kras^{G12D/+}; Pdx1-Cre mice and inhibited the formation of pancreatic cancer precancerous lesions induced by pancreatitis^[9].

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- [3] Su J, Dou Z, Hong H, et al. KRIBB11: A promising drug that promotes microglial process elongation and suppresses neuroinflammation[J]. Frontiers in Pharmacology, 2022, 13: 857081.
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- [7] Huang C, Lu X, Tong L, et al. Requirement for endogenous heat shock factor 1 in inducible nitric oxide synthase induction in murine microglia[J]. Journal of Neuroinflammation, 2015, 12(1): 189.
- [8] Viana P H L, Schvarcz C A, Danics L O, et al. Heat shock factor 1 inhibition enhances the effects of modulated electro hyperthermia in a triple negative breast cancer mouse model[J]. Scientific Reports, 2024, 14(1): 8241.
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