BMS-3

Cat. No.: HY-18304

CAS No.: 1338247-30-5

Molecular Formula: $C_{17}H_{12}Cl_2F_2N_4OS$

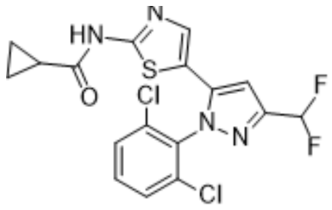
Molecular Weight: 429.27

Target: LIM Kinase (LIMK)

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 : ≥ 100 mg/mL (232.95 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.3295 mL	11.6477 mL	23.2954 mL
5 mM	0.4659 mL	2.3295 mL	4.6591 mL
10 mM	0.2330 mL	1.1648 mL	2.3295 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (5.82 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.82 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.82 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BMS-3 is a potent LIMK inhibitor with IC_{50}s of 5 nM and 6 nM for LIMK1 and LIMK2, respectively.

IC_{50} & Target

LIMK1	LIMK2
5 nM (IC_{50})	6 nM (IC_{50})

In Vitro

BMS-3 (Compound 2) causes a dose-dependent reduction in cell count and induces mitotic arrest by increases in total

nuclear DNA intensity and histone H3 phosphorylation after 24 h treatment in A549 human lung cancer cells. BMS-3 inhibits A549 human lung cancer cells with EC_{50} value of 154 nM[1]. BMS-3 is used to demonstrate the direct participation of LIMK1 in the phosphorylation of Cofilin. Inhibition of p-LIMK with 1-50 μM of BMS-3 results in a dose-dependent decrease of p-Cofilin after 10 min incubation in capacitating conditions. As a control, sperm are also incubated for 10 min under non-capacitating conditions which result in low levels of p-Cofilin. In the presence of 1 or 50 μM of BMS-3, actin polymerization levels are significantly lower compared to controls (DMSO). Mouse sperm are incubated under capacitating conditions for 90 min in the presence or absence of increasing concentrations of p-LIMK inhibitor BMS-3 (0, 1, 10 and 50 μM). The increasing concentrations of BMS-3 result in a strong decrease on the percentage of sperm that undergoes acrosomal exocytosis after stimulation with 20 μM of Progesterone[2].

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

PROTOCOL

Kinase Assay [1]

The protein kinase domains of human LIMK1 and LIMK2 are expressed as glutathione S-transferase fusion proteins using the Bac-to-Bac system in Sf9 cells. Compounds 1 to 6 (e.g., BMS-3) are assayed for inhibition of LIMK1 and LIMK2 protein kinase activity by radioactive phosphate incorporation into biotinylated full-length human destrin. Reactions are done with a concentration series of compound in 25 mM HEPES, 100 mM NaCl, 5 mM $MgCl_2$, 5 mM $MnCl_2$, 1 μM total ATP, 83 μg/mL biotinylated destrin, 167 ng/mL glutathione S-transferase-LIMK1, or 835 ng/mL glutathione S-transferase-LIMK2 in a total volume of 60 μL at room temperature for 30 min (LIMK1) or 60 min (LIMK2). Reactions are terminated by addition of 140 μL of 20% TCA/100 mM sodium pyrophosphate, and the precipitates are harvested onto GF/C unifilter plates. The radioactivity incorporated is determined using a TopCount after addition of 35 μL Microscint scintillation fluid[1].

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

CUSTOMER VALIDATION

- Proc Natl Acad Sci U S A. 2022 May 24;119(21):e2119483119.
- Stem Cell Res Ther. 2022 May 7;13(1):189.
- Cell Death Discov. 2022 Apr 4;8(1):155.

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REFERENCES

[1]. Ross-Macdonald P, et al. Identification of a nonkinase target mediating cytotoxicity of novel kinase inhibitors. Mol Cancer Ther. 2008 Nov;7(11):3490-8.

[2]. Romarowski A, et al. PKA-dependent phosphorylation of LIMK1 and Cofilin is essential for mouse sperm acrosomal exocytosis. Dev Biol. 2015 Sep 15;405(2):237-49.

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