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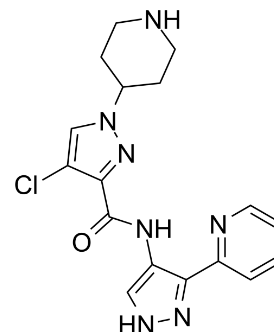
Product Data Sheet

Inhibitors • Screening Libraries • Proteins

BDP5290



Cat. No.:	HY-12437
CAS No.:	1817698-21-7
Molecular Formula:	$C_{17}H_{18}ClN_7O$
Molecular Weight:	371.82
Target:	ROCK
Pathway:	Cell Cycle/DNA Damage; Cytoskeleton; Stem Cell/Wnt; TGF-beta/Smad
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 : 12.5 mg/mL (33.62 mM; Need ultrasonic)

Preparing Stock Solutions	Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
	1 mM	2.6895 mL	13.4474 mL	26.8947 mL
	5 mM	0.5379 mL	2.6895 mL	5.3789 mL
	10 mM	0.2689 mL	1.3447 mL	2.6895 mL

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

BIOLOGICAL ACTIVITY

Description

BDP5290 is a potent inhibitor of both ROCK and MRCK with IC_{50}s of 5 nM, 50 nM, 10 nM and 100 nM for ROCK1, ROCK2, MRCK α and MRCKβ, respectively.

IC_{50} & Target

ROCK1	ROCK2	MRCKα	MRCKβ
5 nM (IC_{50})	50 nM (IC_{50})	10 nM (IC_{50})	100 nM (IC_{50})

In Vitro

The K_i of BDP5290 for MRCKα is 10 nM, which is slightly more than the K_i of 4 nM for MRCKβ. 3 μM BDP5290 completely inhibits myosin II light chain (MLC) phosphorylation induced by MRCKβ, but not by ROCK1 or ROCK2. At higher concentrations, BDP5290 reduces MLC phosphorylation (pMLC) to undetectable levels. BDP5290 reduces MDA-MB-231 invasion at all tested concentrations starting from 0.1 μM, with virtually complete inhibition at 10 μM. After 24 hours in the presence of BDP5290 cell viability is slightly reduced with an EC_{50} >10 μM. Wound closure is inhibited by >60% at 1 μM BDP5290, a concentration that has no effect on cell viability[2].

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

PROTOCOL

Kinase Assay [2]	MRCKα, MRCKβ, ROCK1 and ROCK2 assays are performed using an IMAP fluorescence polarization assay format. 8 to 12 nM of each kinase is incubated for 60 min at room temperature with 100 nM FAM-S6-ribosomal protein derived peptide in the presence of 1 μM ATP and 0.5 mM $MgCl_2$ in 20 mM Tris buffer (pH 7.4) containing 0.01% Tween-20 and 1 mM DTT (MRCKα and β); or 1 μM ATP, 10 mM $MgCl_2$ in 20 mM Tris buffer (pH 7.5) containing 0.25 mM EGTA 0.01% Triton X-100 and 1 mM DTT (ROCK1 and ROCK2). Typically, dose response analysis are performed over concentration ranges from 0.005 to 100 μM. Reactions are stopped by adding 2 assay volumes of 0.25% (v/v) IMAP binding reagent in 1×IMAP binding buffer. After 30 min incubation to allow binding reagent to bind phosphorylated peptide, fluorescence polarization is measured on a plate reader at excitation (470 nm) and emission (530 nm) wavelengths. Inhibition is calculated using no inhibitor and no enzyme controls as 0 and 100% inhibition, respectively. Kinase selectivity profiling is performed by Eurofins with 10 μM ATP and 10 μ M BDP5290[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [2]	MDA MB 231 or SCC12 cells are plated in a 96 well plate and cultured for 24 hours. Cells are then cultured for 24 hours in SCC12 medium with DMSO vehicle or indicated concentrations of BDP5290 in an IncuCyte ZOOM. Pictures are taken every 3 hours and confluence is measured using the IncuCyte analysis software. AlamarBlue is added to the medium and the cells are cultured for an additional day. Absorbances at 570 nm and at 600 nm are measured to assess cell health[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Gandalovi?ová A, et al. Migrastatics-Anti-metastatic and Anti-invasion Drugs: Promises and Challenges. Trends Cancer. 2017 Jun;3(6):391-406.

[2]. Unbekandt M, et al. A novel small-molecule MRCK inhibitor blocks cancer cell invasion. Cell Commun Signal. 2014 Oct 5;12:54.

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

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