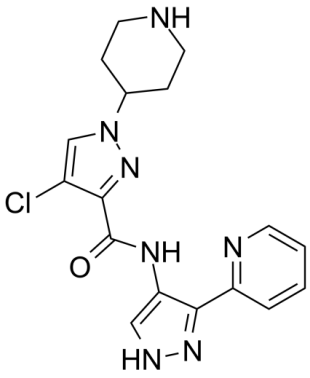
Data Sheet

Product Name:	BDP5290
Cat. No.:	CS-8133
CAS No.:	1817698-21-7
Molecular Formula:	C17H18ClN7O
Molecular Weight:	371.82
Target:	ROCK
Pathway:	Cell Cycle/DNA Damage; Stem Cell/Wnt; TGF-beta/Smad
Solubility:	DMSO : 12.5 mg/mL (33.62 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

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**, **MRCKα** and **MRCKβ**, respectively. IC50 & Target: IC50: 5 nM (ROCK1), 50 nM (ROCK2), 10 nM (MRCKα), 100 nM (MRCKβ)[1] **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].

PROTOCOL (Extracted from published papers and Only for reference)

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 μM BDP5290[2]. **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].

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.

CAIndexNames:

1H-Pyrazole-3-carboxamide, 4-chloro-1-(4-piperidinyl)-N-[3-(2-pyridinyl)-1H-pyrazol-4-yl]-

SMILES:

ClC(C(C(NC1=CNN=C1C2=NC=CC=C2)=O)=N3)=CN3C4CCNCC4

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

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