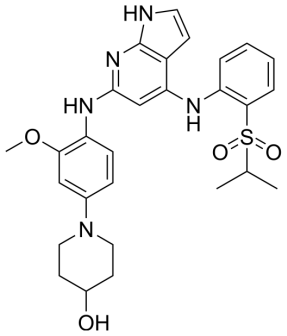
Mps1-IN-1



Cat. No.:	HY-13298
CAS No.:	1125593-20-5
Molecular Formula:	$C_{28}H_{33}N_5O_4S$
Molecular Weight:	535.66
Target:	Mps1
Pathway:	Cell Cycle/DNA Damage; Cytoskeleton
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 : ≥ 39 mg/mL (72.81 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.8669 mL	9.3343 mL	18.6686 mL
5 mM	0.3734 mL	1.8669 mL	3.7337 mL
10 mM	0.1867 mL	0.9334 mL	1.8669 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 (4.67 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Mps1-IN-1 is a potent, selective and ATP-competitive Mps1 kinase inhibitor, with an IC_{50} and a K_d of 367 nM and 27 nM[1].

IC_{50} & Target

Mps1 367 nM (IC_{50})	Mps1 27 nM (Kd)	ALK 21 nM (Kd)	LTK 29 nM (Kd)
PYK2 280 nM (Kd)	FAK 440 nM (Kd)	IGF1R 750 nM (Kd)	INSR 470 nM (Kd)
CLK1 1900 nM (Kd)	ERK2 2900 nM (Kd)	INSRR 1200 nM (Kd)	TNK1 2600 nM (Kd)
TNK2	GAK		

	3100 nM (Kd)	1100 nM (Kd)
In Vitro	Mps1-IN-1 is a potent, selective and ATP-competitive Mps1 kinase inhibitor, with an IC_{50} and a K_d of 367 nM and 27 nM. Mps1-IN-1 also has high affinity for ALK, and LTK, with K_ds of 21 and 39 nM, respectively, but shows little or no inhibition on other 352 member kinases. Mps1-IN-1 (5, 10 μM) induces bypass of a checkpoint-mediated mitotic arrest in U2OS cells. Mps1-IN-1 disrupts recruitment of Mad2 to kinetochores, and reduces the phosphorylation status of Aurora B at threonine-232 (Thr232). Mps1-IN-1 (10 μM) shows no effect on centrosome duplication. In addition, Mps1-IN-1 (5-10 μM) suppresses the proliferative capacity of HCT116[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.	

PROTOCOL

Kinase Assay [1]	The kinase binding assay is used to assess compound binding to TTK by monitoring displacement of a fluorescently labeled, ATP site-directed kinase inhibitor (Kinase Tracer 236) from the kinase active site. Each 15 μL assay contains 5 nM TTK, variable amounts of test compound (Mps1-IN-1), 30 nM Kinase Tracer 236, 2 nM Eu-anti-GST Antibody, and 1% DMSO (residual from compound dilution) in Kinase Buffer A (50 mM HEPES pH 7.5, 10 mM $MgCl_2$, 1 mM EGTA, 0.01% Brij-35). Binding assays are initiated by addition of 5 μL of test compound (from 2-fold dilution series) to 5 μL of a kinase/antibody mixture, followed by addition of 5 μL of antibody. Assay plates are read using using standard Eu-based TR-FRET settings with excitation at 340 nm and emission monitored at 615 nm (donor) and 665 nm (acceptor). Emission intensities are measured over a 200 μs window following a 100 μs post-excitation delay[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [1]	U2OS cells expressing doxycycline-inducible PLK4 are plated in 96 well plates. A double thymidine block is performed using the following treatment regimen: thymidine for 18-20 hrs., release for 10 hrs. with doxycycline induction of PLK4 during this time, then a second thymidine block, followed by release. Six hours after the 2nd thymidine release, Mps1-IN-1 (or DMSO vehicle) is added and the proliferation of the cell populations is monitored with Cell Titer GLO assay[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Cell Biochem. 2021 Feb;122(2):290-300.
- J Cancer. 2023 May 21; 14(9): 1515-1530.
- bioRxiv. 2021 Feb 26.

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REFERENCES

[1]. Kwiatkowski N, et al. Small-molecule kinase inhibitors provide insight into Mps1 cell cycle function. Nat Chem Biol. 2010 May;6(5):359-68.

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