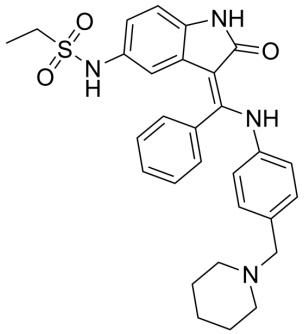
Hesperadin



Cat. No.:	HY-12054
CAS No.:	422513-13-1
Molecular Formula:	$C_{29}H_{32}N_4O_3S$
Molecular Weight:	516.65
Target:	Aurora Kinase; Autophagy; Influenza Virus; Parasite
Pathway:	Cell Cycle/DNA Damage; Epigenetics; Autophagy; Anti-infection
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 (193.55 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.9355 mL	9.6777 mL	19.3555 mL
	5 mM	0.3871 mL	1.9355 mL	3.8711 mL
	10 mM	0.1936 mL	0.9678 mL	1.9355 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 (4.84 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.84 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Hesperadin is an ATP competitive indolinone inhibitor of Aurora A and B. Hesperadin inhibits Aurora B with an IC_{50} of 250 nM. Hesperadin inhibits the growth of Trypanosoma brucei by blocking nuclear division and cytokinesis. Hesperadin also is a broad-spectrum influenza antiviral[1][2][3].

IC_{50} & Target		
	Trypanosoma	Aurora B 250 nM (IC_{50})

In Vitro

Hesperadin (10-100 nM) inhibits the Aurora kinase-1 (TbAUK1)-mediated phosphoryation of trypanosome histone H3 (TbH3) in a dose dependent manner, with an IC_{50} of 40 nM[1].

Hesperadin (0.01-10 μM; 24 or 48 hours) inhibits growth of bloodstream forms (BF) and procyclic forms (PF) cultures[1]. Hesperadin (100-200 nM; 24-72 hours) alters cell morphology and inhibits cell cycle progression similar to the RNAi knockdown of TbAUK1[1].

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

Cell Viability Assay[1]

Cell Line:	M110 cells
Concentration:	0.01, 0.1, 1, 10 μM
Incubation Time:	24 hours or 48 hours
Result:	Inhibiting growth of BF cultures with IC_{50} of 50 nM, while the inhibition of PF growth required approximately 11-fold more Hesperadin, with IC_{50} of 550 nM.

Cell Cycle Analysis[1]

Cell Line:	M110 cells
Concentration:	100, 200 nM
Incubation Time:	24, 48, 72 hours
Result:	Had a strong effect on cell growth and mitotic progression at 100-200 nM.

In Vivo

Hesperadin (20 mg/kg/d; i.v.) prolongs the survival of xenograft mice via synergistic effect with temozolomide (TMZ)[2].

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Animal Model:	6-week-old female nude mice injected GBM cells[2]
Dosage:	20 mg/kg/d
Administration:	I.v. injection
Result:	Increased the survival of xenograft mice models.

CUSTOMER VALIDATION

- Adv Sci (Weinh). 2022 Oct 30;e2205091.
- Sci Rep. 2021 Jan 27;11(1):2283.
- Brain Res Bull. 2021 Sep 14;S0361-9230(21)00269-0.
- Exp Cell Res. 2021 Jul 21;112741.
- Behav Neurol. 2020 Feb 3;2020:2476861.

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REFERENCES

[1]. Neal J, et, al. The cell cycle as a therapeutic target against Trypanosoma brucei: Hesperadin inhibits Aurora kinase-1 and blocks mitotic progression in bloodstream forms. Mol Microbiol. 2009 Apr; 72(2): 442-58.

[2]. Wahafu A, et, al. Targeting Aurora kinase B attenuates chemoresistance in glioblastoma via a synergistic manner with temozolomide. Pathol Res Pract. 2019 Nov;

215(11): 152617.

[3]. Hu Y, et, al. Chemical Genomics Approach Leads to the Identification of Hesperadin, an Aurora B Kinase Inhibitor, as a Broad-Spectrum Influenza Antiviral. Int J Mol Sci. 2017 Sep 8;18(9):1929.

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