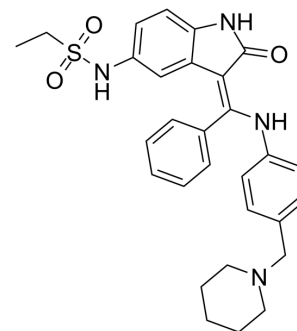


Hesperadin

Cat. No.:	HY-12054		
CAS No.:	422513-13-1		
Molecular Formula:	C ₂₉ H ₃₂ N ₄ O ₃ S		
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	6 months
		-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (193.55 mM)
 * "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		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

- 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
- 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 IC₅₀ 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]}.

IC₅₀ & Target

Trypanosoma	Aurora B 250 nM (IC ₅₀)
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In Vitro

Hesperadin (10-100 nM) inhibits the Aurora kinase-1 (TbAUK1)-mediated phosphorylation of trypanosome histone H3 (TbH3) in a dose dependent manner, with an IC₅₀ of 40 nM^[1].

Hesperadin (0.01-10 μ M; 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 μ M
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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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

- Sci Rep. 2021 Jan 27;11(1):2283.
- Exp Cell Res. 2021 Jul 21;112741.
- Brain Res Bull. 2021 Sep 14;S0361-9230(21)00269-0.
- Behav Neurol. 2020 Feb 3;2020:2476861.
- Research Square Preprint. 2021 Jan.

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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.

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

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