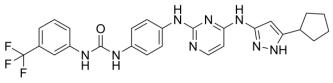
CD532

Cat. No.:	HY-112273
CAS No.:	1639009-81-6
Molecular Formula:	$C_{26}H_{25}F_3N_8O$
Molecular Weight:	522.52
Target:	Aurora Kinase
Pathway:	Cell Cycle/DNA Damage; Epigenetics
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 : 250 mg/mL (478.45 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.9138 mL	9.5690 mL	19.1380 mL
	5 mM	0.3828 mL	1.9138 mL	3.8276 mL
	10 mM	0.1914 mL	0.9569 mL	1.9138 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: ≥ 2.08 mg/mL (3.98 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (3.98 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (3.98 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CD532 is a potent Aurora A kinase inhibitor with an IC_{50} of 45 nM. CD532 has the dual effect of blocking Aurora A kinase activity and driving degradation of MYCN. CD532 also can directly interact with AURKA and induces a global conformational shift. CD532 can be used for the research of cancer[1][2].

IC_{50} & Target

Aurora A
45 nM (IC_{50})

In Vitro

CD532 (1-10000 nM; 72 h) is cytotoxic in MYCN-amplified neuroblastoma cell lines SK-N-BE(2) and Kelly, with EC_{50}s of 223.2 nM and 146.7 nM, respectively[1].
CD532 (0.1-1 μM; 24 h) causes dose-dependent loss of MYCN protein in SK-N-BE(2) cells[1].
CD532 (1 μM; 6 h) prevents S-phase entry in SK-N-BE(2) cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	SK-N-BE(2) and Kelly cells
Concentration:	1, 10, 100, 1000, 10000 nM
Incubation Time:	72 hours
Result:	Inhibited the cell viability of SK-N-BE(2) and Kelly cells, with EC_{50}s of 223.2 nM and 146.7 nM, respectively.

Cell Cycle Analysis[1]

Cell Line:	SK-N-BE(2) cells
Concentration:	1 μM
Incubation Time:	4 hours
Result:	Resulted in a rapid and potent loss of S-phase and accumulation in both G0/G1 and G2.

Western Blot Analysis[1]

Cell Line:	SK-N-BE(2) cells
Concentration:	0.1, 0.25, 0.5, 1 μM
Incubation Time:	2, 4, 6, 24 hours
Result:	Causes dose-dependent and time-dependent loss of MYCN protein.

In Vivo

CD532 (25 mg/kg; i.p. twice weekly for 3 weeks) decreases the tumor volume and increases survival in mice with subcutaneous sonic hedgehog (SHH)-subtype medulloblastoma[1].
CD532 (60 mg/kg; i.p. for 2 days) decreases the level of MYCN protein in MYCN-amplified neuroblastoma xenografts[1].
CD532 (20 mg/kg; i.p.) shows a serum half-life of ~1.5 hours and AUC0-24 of 27 μM•h in mice[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Homozygous nu/nu mice with SHH-subtype MYCN-expressing medulloblastoma[1]
Dosage:	25 mg/kg
Administration:	I.p. twice weekly for 3 weeks
Result:	Decreased the level of MYCN protein and tumor volume and increases survival.

REFERENCES

[1]. Gustafson WC, et, al. Drugging MYCN through an allosteric transition in Aurora kinase A. Cancer Cell. 2014 Sep 8;26(3):414-427.

[2]. Lee JK, et, al. N-Myc Drives Neuroendocrine Prostate Cancer Initiated from Human Prostate Epithelial Cells. Cancer Cell. 2016 Apr 11;29(4):536-547.

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

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