CLK-IN-T3

Cat. No.: HY-115470

CAS No.: 2109805-56-1

Molecular Formula: $C_{28}H_{30}N_6O_2$

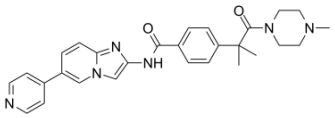
Molecular Weight: 482.58

Target: CDK; DYRK

Pathway: Cell Cycle/DNA Damage; Protein Tyrosine Kinase/RTK

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 : 4.83 mg/mL (10.01 mM; Need ultrasonic and warming)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0722 mL	10.3610 mL	20.7220 mL
	5 mM	0.4144 mL	2.0722 mL	4.1444 mL
	10 mM	0.2072 mL	1.0361 mL	2.0722 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 (4.31 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.31 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CLK-IN-T3 is a high potent, selective, and stable CDC-like kinase (CLK) inhibitor with IC_{50}s of 0.67 nM, 15 nM, and 110 nM for CLK1, CLK2, and CLK3 protein kinases, respectively. CLK-IN-T3 has anti-cancer activity[1].

IC_{50} & Target

CLK1 0.67 nM (IC_{50})	CLK2 15 nM (IC_{50})	CLK3 110 nM (IC_{50})	DYRK1A 260 nM (IC_{50})
DYRK1B 230 nM (IC_{50})			

In Vitro

CLK-IN-T3 inhibits DYRK1A (IC_{50}=260 nM) and DYRK1B (IC_{50}=230 nM)[1].

CLK-IN-T3 (0.1-10.0 μM; 24 hours) results in mild cell cycle arrest at the G2/M boundary with long-duration (24 h)[1].
CLK-IN-T3 (0.5-1.0 μM; 6 hours) decreases phosphorylation of CLK-targeted SR proteins and CLK proteins increase slightly[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cycle Analysis[1]

Cell Line:	HCT-116 cells
Concentration:	0.1, 0.5, 1.0, 5.0, 10.0 μM
Incubation Time:	24 hours
Result:	Resulted in mild cell cycle arrest at the G2/M boundary with long-duration (24 h).

Western Blot Analysis[1]

Cell Line:	HCT-116 cells
Concentration:	0.5, 1.0 μM
Incubation Time:	6 hours
Result:	Decreased phosphorylation of CLK-targeted SR proteins and CLK proteins increased slightly.

REFERENCES

[1]. Funnell T, et al. CLK-dependent exon recognition and conjoined gene formation revealed with a novel smallmolecule inhibitor. Nat Commun. 2017 Feb 23;8(1):7.

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