NU6102

Cat. No.: HY-15569

CAS No.: 444722-95-6

Molecular Formula: $C_{18}H_{22}N_6O_3S$

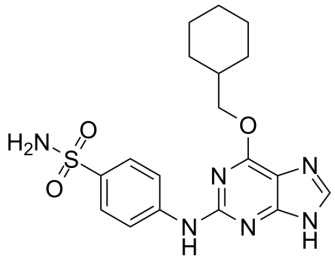
Molecular Weight: 402.47

Target: CDK

Pathway: Cell Cycle/DNA Damage

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 (248.47 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4847 mL	12.4233 mL	24.8466 mL
5 mM	0.4969 mL	2.4847 mL	4.9693 mL
10 mM	0.2485 mL	1.2423 mL	2.4847 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 (6.21 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.21 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.21 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

NU6102 is a potent CDK1 and CDK2 inhibitor with IC_{50}s of 9.5 nM and 5.4 nM for CDK1/cyclinB and CDK2/cyclinA3, respectively. NU6102 shows selectivity for CDK1/CDK2 over CDK4 (IC_{50} of 1.6 μM), DYRK1A (IC_{50} of 0.9 μM), PDK1 (IC_{50} of 0.8 μM) and ROCKII (IC_{50} of 0.6 μM)[1][2].

IC_{50} & Target

Cdk1/cyclin B 9.5 nM (IC_{50})	CDK2/cyclin A3 5.4 nM (IC_{50})	CDK4 1.6 μM (IC_{50})	DYRK1A 0.9 μM (IC_{50})
PDK1			

	0.8 μM (IC_{50})
In Vitro	NU6102 (0-30 μM; 1-24 hours; SKUT 1B cells) treatment induces a G2 arrest, inhibition of Rb phosphorylation and cytotoxicity (LC_{50} 2.6 μM for a 24 h exposure) in SKUT-1B cells[3]. NU6102 inhibits cell growth and causes cell cycle phase arrest in human breast cancer cell lines, G2/M arrest in asynchronously growing cell lines and G1/S arrest in cells released from serum starvation, and in Xenopus nuclei in a timedependent manner[3]. NU6102 selectively inhibits the growth of CDK2 WT (wild type) versus KO MEFs (knockout mouse embryo fibroblasts) (GI_{50} of 14 μM versus >30 μM)[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only. Cell Cycle Analysis[3]

Cell Line:	SKUT 1B cells
Concentration:	0 μM, 3 μM, 10 μM, and 30 μM
Incubation Time:	1 hours, 3 hours, 6 hours, and 24 hours
Result:	Induced a G2 arrest, inhibition of Rb phosphorylation and cytotoxicity (LC_{50} 2.6 μM for a 24 h exposure).

In Vivo	The pharmacokinetics of NU6102 is determined following i.v. and i.p. administration in Balb/C mice. The limited solubility of NU6102 meant the maximum administrable dose is 1 mg/kg i.v. and 10 mg/kg i.p. NU6102 is liberated following either i.p. or i.v. administration of NU6301, and following i.v. administration peak plasma levels of 12 μM NU6102 is observed 5 min post administration, whereas following administration of the maximum administrable dose of NU6102 i.v. the peak concentration achieved is 0.92 μM. The plasma half-life of NU6102 liberated following administration of NU6301 is 42 min following i.p. and 10 min following i.v. administration[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Ian R Hardcastle, et al. N2-substituted O6-cyclohexylmethylguanine derivatives: potent inhibitors of cyclin-dependent kinases 1 and 2. J Med Chem. 2004 Jul 15;47(15):3710-22.

[2]. David J Pratt, et al. Dissecting the determinants of cyclin-dependent kinase 2 and cyclin-dependent kinase 4 inhibitor selectivity. J Med Chem. 2006 Sep 7;49(18):5470-7.

[3]. Huw D Thomas , et al. Preclinical in vitro and in vivo evaluation of the potent and specific cyclin-dependent kinase 2 inhibitor NU6102 and a water soluble prodrug NU6301. Eur J Cancer. 2011 Sep;47(13):2052-9.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA