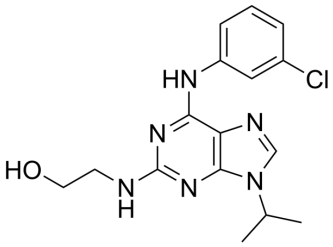
NG 52

Cat. No.:	HY-15154
CAS No.:	212779-48-1
Molecular Formula:	$C_{16}H_{19}ClN_6O$
Molecular Weight:	346.81
Target:	CDK
Pathway:	Cell Cycle/DNA Damage
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 : 75 mg/mL (216.26 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8834 mL	14.4171 mL	28.8342 mL
	5 mM	0.5767 mL	2.8834 mL	5.7668 mL
	10 mM	0.2883 mL	1.4417 mL	2.8834 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 (7.21 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.21 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

NG 52 is a potent, cell-permeable, selective, ATP-compatible and orally active Cdc28p and Pho85p kinase inhibitor with IC_{50}s of 7 μM and 2 μM, respectively. NG 52 also inhibits the activity of phosphoglycerate kinase 1 (PGK1) with an IC_{50} of 2.5 μM. NG 52 is inactive against yeast kinases Kin28p, Srb10, and Cak1p[1][2].

IC_{50} & Target

cdc2-cyclin B 0.34 μM (IC_{50})	Pho85p 2 nM (IC_{50})	Cdc28p 7 μM (IC_{50})	Phosphoglycerate kinase 1 (PGK1) 2.5 μM (IC_{50})

In Vitro

NG 52 (Compound 52) inhibits growth in a drug-sensitized yeast strain (S. cerevisiae) with a GI_{50} of 30 μM. NG 52 is active against cdc2-cyclin B with an IC_{50} value of 340 nM[1].

NG 52 dose-dependently inhibits the proliferation of glioma U87 and U251 cell lines with GI_{50} values of 7.8 μM and 5.2 μM, respectively, meanwhile it potently inhibits the proliferation of primary glioma cells[2].

NG 52 (12.5-50 μM) effectively inhibits the phosphorylation of PDHK1 at Thr338 site and the phosphorylation of PDH at Ser293 site in U87 and U251 cells, resulting in more pyruvic acid entering the Krebs cycle with increased production of ATP and ROS[2].

NG 52 can reverse the Warburg effect by enhancing the activity of pyruvate dehydrogenase (PDH) through inhibiting the activity of PGK1, and switched cellular glucose metabolism from anaerobic mode to aerobic mode[2].

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

Cell Proliferation Assay[2]

Cell Line:	Glioma U87 and U251 cells
Concentration:	0 μM, 12.5 μM, 25 μM, 50 μM
Incubation Time:	6 days
Result:	Potently inhibited the proliferation of primary glioma cells.

Western Blot Analysis[2]

Cell Line:	Glioma U87 and U251 cells
Concentration:	0 μM, 12.5 μM, 25 μM, 50 μM
Incubation Time:	12 hours or 24 hours
Result:	Potently inhibited the proliferation of primary glioma cells.

In Vivo

NG 52 (50-150 mg/kg; oral administration; daily; for 13 days) treatment dose-dependently suppresses the growth of glioma xenograft[2].

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

Animal Model:	Female nu/nu mice (5-week-old) injected with glioma cells[2]
Dosage:	50 mg/kg, 100 mg/kg, 150 mg/kg
Administration:	Oral administration; daily; for 13 days
Result:	Dose-dependently suppressed the growth of glioma xenograft.

CUSTOMER VALIDATION

- Acta Pharmacol Sin. 2020 Jul 31.
- Food Chem X. 2024 Jan 9, 101125.
- J Appl Physiol (1985). 2023 Jun 29.

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REFERENCES

[1]. ray NS, Wodicka L, Thunnissen AM et al. Exploiting chemical libraries, structure, and genomics in the search for kinase inhibitors. Science. 1998 Jul 24;281(5376):533-8.

[2]. Wen-Liang Wang, et al. Pharmacologically inhibiting phosphoglycerate kinase 1 for glioma with NG52. Acta Pharmacol Sin. 2020 Jul 31.

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