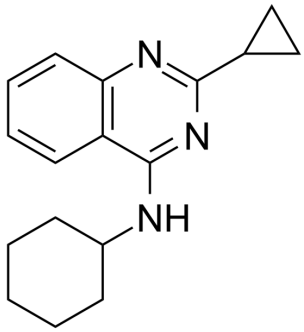
TH10785

Cat. No.:	HY-147313
CAS No.:	1002801-51-5
Molecular Formula:	$C_{17}H_{21}N_3$
Molecular Weight:	267.37
Target:	Others
Pathway:	Others
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (374.01 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.7401 mL	18.7007 mL	37.4014 mL
	5 mM	0.7480 mL	3.7401 mL	7.4803 mL
	10 mM	0.3740 mL	1.8701 mL	3.7401 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 (9.35 mM); Suspended solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (9.35 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.35 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

TH10785 is a DNA glycosylase 1 (OGG1) activator, TH10785 can interact with the phenylalanine-319 and glycine-42 amino acids of OGG1 and increase the enzyme activity, generates β, δ-lyase enzymatic function. TH10785 can control the catalytic activity mediated by a nitrogen base within its molecular structure. TH10785 can be used for the research of various diseases and aging connected with DNA oxidative lesions[1].

IC_{50} & Target

OGG1 (KD=5.5 μM)[1]

In Vitro

TH10785 (6.25 μM, 30 min) induces a de novo β, δ-elimination in vitro, allowing for AP sites as new substrates[1].
TH10785 (10 μM, 0-2 min) allows OGG1 to increase DNA repair by addressing AP sites[1].
TH10785 (0-20 μM, 72 h) induces OGG1 β, δ-lyase activity shifts cells toward PNKP1 dependence[1].
TH10785 (2 μM) has affinity to OGG1 (KD=5.5 μM) increased when adding an AP site analog containing double-stranded DNA (KD=1.3 μM)[1].

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

Cell Viability Assay[1]

Cell Line:	U2OS cells
Concentration:	0-20 μM
Incubation Time:	72 hours
Result:	Reduced combination with PNKP1 inhibition for TH10785.

Western Blot Analysis[1]

Cell Line:	U2OS cells
Concentration:	0.65 μM; 10 μM
Incubation Time:	30 min; 24 h
Result:	Caused an up-regulation of members of the DDR through β, δ-elimination in combination with PNKP1 inhibition and APE1-independent de novo β, δ-elimination of AP sites by OGG1 in the presence of TH10785.

RT-PCR[1]

Cell Line:	U2OS cells
Concentration:	10 μM
Incubation Time:	1 h
Result:	Decreased oxidative damage in guanine-rich regions of the genome.

Immunofluorescence[1]

Cell Line:	U2OS OGG1-GFP cells
Concentration:	1 μM
Incubation Time:	0-2 min
Result:	Ecruited more OGG1 to laser-damaged sites.

REFERENCES

[1]. Maurice Michel, et al. Small-molecule activation of OGG1 increases oxidative DNA damage repair by gaining a new function. Science. 2022 Jun 24;376(6600):1471-1476.

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

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