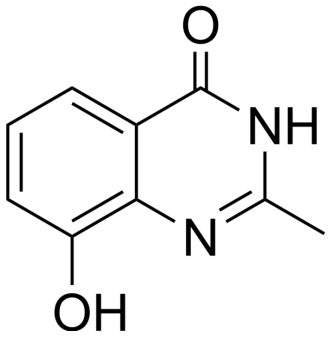
NU1025

Cat. No.:	HY-15044
CAS No.:	90417-38-2
Molecular Formula:	$C_9H_8N_2O_2$
Molecular Weight:	176.17
Target:	PARP
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 : 100 mg/mL (567.63 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	5.6763 mL	28.3817 mL	56.7634 mL
	5 mM	1.1353 mL	5.6763 mL	11.3527 mL
	10 mM	0.5676 mL	2.8382 mL	5.6763 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 (11.81 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (11.81 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

NU1025 is a potent PARP inhibitor with an IC_{50} of 400 nM and a K_i of 48 nM. NU1025 potentiates the cytotoxicity of ionizing radiation and anticancer agents. NU1025 has anti-cancer and neuroprotective activity[1][2][3].

IC_{50} & Target

IC50: 400 nM (PARP)[2]
Ki: 48 nM (PARP)[3]

In Vitro

NU1025 (0.2 mM) pretreatment restores cell viability to approximately 73% and 82% in H_2O_2 and SIN-1 injured cells, respectively[1].
NU1025 enhances the cytotoxicity of the DNA-methylating agent MTIC, γ-irradiation and bleomycin 3.5-, 1.4- and 2-fold respectively in L1210 cells. The recovery from potentially lethal γ-irradiation damage cytotoxicity in plateau-phase cells is

also inhibited by NU 1025. NU1025 causes a marked retardation of DNA repair[2].

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

Cell Viability Assay[1]

Cell Line:	PC12 cells
Concentration:	0.2 mM
Incubation Time:	6.5 hours
Result:	Restored cell viability to approximately 73% and 82% in H_2O_2 and SIN-1 injured cells.

In Vivo

NU1025 (1-3 mg/kg; intraperitoneal injection; male Sprague Dawley rats) treatment at 1 and 3 mg/kg reduces total infarct volume to 25% and 45%, respectively, when administered 1 h before reperfusion. NU1025 also produces significant improvement in neurological deficits. Neuroprotection with NU1025 is associated with reduction in PAR accumulation, reversal of brain NAD depletion and reduction in DNA fragmentation[1].

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

Animal Model:	Male Sprague Dawley rats (250-270 g) induced focal cerebral ischemia[1]
Dosage:	1 mg/kg, 3 mg/kg
Administration:	Intraperitoneal injection
Result:	At 1 and 3 mg/kg, reduced total infarct volume to 25% and 45%, respectively.

REFERENCES

[1]. Kaundal RK, et al. Neuroprotective effects of NU1025, a PARP inhibitor in cerebral ischemia are mediated through reduction in NAD depletion and DNA fragmentation. Life Sci. 2006 Nov 10;79(24):2293-302.

[2]. Bowman KJ, et al. Potentiation of anti-cancer agent cytotoxicity by the potent poly(ADP-ribose) polymerase inhibitors NU1025 and NU1064. Br J Cancer. 1998 Nov;78(10):1269-77.

[3]. Delaney CA, et al. Potentiation of temozolomide and topotecan growth inhibition and cytotoxicity by novel poly(adenosine diphosphoribose) polymerase inhibitors in a panel of human tumor cell lines. Clin Cancer Res. 2000 Jul;6(7):2860-7.

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