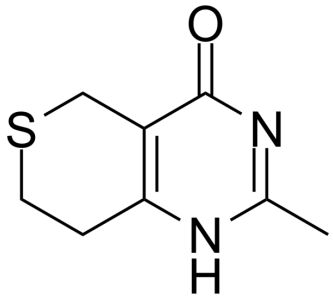
DR2313

Cat. No.:	HY-105692
CAS No.:	284028-90-6
Molecular Formula:	$C_8H_{10}N_2OS$
Molecular Weight:	182.24
Target:	PARP
Pathway:	Cell Cycle/DNA Damage; Epigenetics
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 100 mg/mL (548.73 mM)
DMSO : 12.5 mg/mL (68.59 mM; Need ultrasonic)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.4873 mL	27.4363 mL	54.8727 mL
5 mM	1.0975 mL	5.4873 mL	10.9745 mL
10 mM	0.5487 mL	2.7436 mL	5.4873 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

DR2313 is a potent, selective, competitive and brain-penetrant inhibitor of poly(ADP-ribose) polymerase (PARP), with IC_{50}s of 0.20 μM and 0.24 μM for PARP-1 and PARP-2, respectively. DR2313 exhibits neuroprotective effects on ischemic injuries in vitro and in vivo[1][2].

IC_{50} & Target

PARP-1	PARP-2
0.20 μM (IC_{50})	0.24 μM (IC_{50})

In Vitro

DR2313 (0.016-16.4 μM; 30 min) inhibits poly(ADP-ribosyl)ation reaction in nuclear extracts of rat brain, with a K_i of 0.23 μM[1].

DR2313 shows more powerful inhibition of the poly(ADP-ribosyl)ation in the nuclear extracts of the rat brain (IC_{50}=0.20 μM) than 3AB (35.4 μM), PND (0.56 μM), DIQ (2.96 μM), and DPQ (0.96 μM)[1].

DR2313 (1-100 μM; 10 min) shows a weak inhibition of the mono(ADP-ribosyl)ation in a concentration-dependent manner (IC_{50}=59 μM)[1].

DR2313 (0.1-30 μM; pretreated for 30 min) reduces hydrogen peroxide (500 μM; 4 h) or glutamate (1 mM; 30 min) induced excessive formation of poly(ADP-ribose) and cell death[1].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	DR2313 (3-10 mg/kg i.v. bolus or infusion for 6 h) significantly reduces the cortical infarct volume in both permanent and transient focal ischemia models in rats[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Wistar rats (220-300 g) with permanent MCA occlusions (pMCAos) and transient MCA occlusions (tMCAos)[1]
Dosage:	3, 10 mg/kg
Administration:	I.v. bolus and i.v. infusion for 6 h beginning 5 min before the onset of ischemia
Result:	Reduced the infarct volume in a dose-dependent manner in pMCAo and tMCAo model.

REFERENCES

[1]. Nakajima H, et, al. A newly synthesized poly(ADP-ribose) polymerase inhibitor, DR2313 [2-methyl-3,5,7,8-tetrahydrothiopyrano[4,3-d]-pyrimidine-4-one]: pharmacological profiles, neuroprotective effects, and therapeutic time window in cerebral ischemia in r

[2]. Xu Z, et, al. Endonuclease G does not play an obligatory role in poly(ADP-ribose) polymerase-dependent cell death after transient focal cerebral ischemia. Am J Physiol Regul Integr Comp Physiol. 2010 Jul;299(1):R215-21.

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