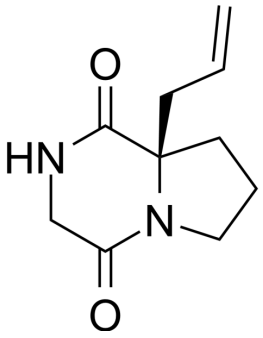
NNZ 2591

Cat. No.:	HY-148195
CAS No.:	847952-38-9
Molecular Formula:	$C_{10}H_{14}N_2O_2$
Molecular Weight:	194.23
Target:	Others
Pathway:	Others

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 : 18.75 mg/mL (96.54 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	5.1485 mL	25.7427 mL	51.4854 mL
	5 mM	1.0297 mL	5.1485 mL	10.2971 mL
	10 mM	0.5149 mL	2.5743 mL	5.1485 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: ≥ 1.5 mg/mL (7.72 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 1.5 mg/mL (7.72 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1.5 mg/mL (7.72 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

NNZ 2591 is a synthetic analogue of a small peptide of cyclic glycine proline (cGP). NNZ 2591 shows orally active and cross the blood-brain barrier. NNZ 2591 shows neuroprotective after ischemic brain injury. NNZ 2591 improves motor function in a rat model of Parkinson's disease. NNZ 2591 has the potential for the research of ischemic brain injury and angelman syndrome[1][2][3].

In Vivo

NNZ 2591 (30 mg/kg; p.o.) prevented scopolamine-induced memory impairment in rats[1].
NNZ 2591 (2, 20, 100 ng/rat; i.c.v.) shows neuroprotection in rats[2].

NNZ 2591 (3 mg/kg; s.c.; daily for 5 days) completely preventes brain damage and significantly reduces the L/R ratio of time taken to touch to the patch at 5 d after injury in rats[2].

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

Animal Model:	4 months, male young adult Wistar rats[1].
Dosage:	30 mg/kg
Administration:	P.o.; 10 min after the (scopolamine) i.p. administration.
Result:	Significantly reduced the number of M2AchR positive neurons, significantly reduced the density of synaptophysin in the CA3 and CA4 sub-regions, and altered TH terminal staining in the striatum.

Animal Model:	280-310 g adult male Wistar rats[2].
Dosage:	2, 20, 100 ng/rat
Administration:	I.c.v.; 2 h after HI injury
Result:	Reduced overall tissue damage in the sub-regions of the hippocampus, DG, cerebral cortex and the striatum.

Animal Model:	280-310 g adult male Wistar rats[2].
Dosage:	3 mg/kg
Administration:	S.c.; daily for 5 days
Result:	Significantly reduced the median of tissue damage scores in the CA1-2, CA3 and CA4 sub-regions of the hippocampus, the DG.

REFERENCES

[1]. Guan J, et al. NNZ-2591, a novel diketopiperazine, prevented scopolamine-induced acute memory impairment in the adult rat. Behav Brain Res. 2010 Jul 11;210(2):221-8.

[2]. Guan J, et al. Peripheral administration of a novel diketopiperazine, NNZ 2591, prevents brain injury and improves somatosensory-motor function following hypoxia-ischemia in adult rats. Neuropharmacology. 2007 Nov;53(6):749-62.

[3]. Copping NA, et al. Emerging Gene and Small Molecule Therapies for the Neurodevelopmental Disorder Angelman Syndrome. Neurotherapeutics. 2021 Jul;18(3):1535-1547.

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