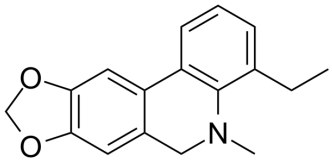
HLY78

Cat. No.:	HY-122816
CAS No.:	854847-61-3
Molecular Formula:	$C_{17}H_{17}NO_2$
Molecular Weight:	267.32
Target:	Wnt; β-catenin; Apoptosis
Pathway:	Stem Cell/Wnt; Apoptosis
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 : 26 mg/mL (97.26 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.7408 mL	18.7042 mL	37.4083 mL
5 mM	0.7482 mL	3.7408 mL	7.4817 mL
10 mM	0.3741 mL	1.8704 mL	3.7408 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 (7.78 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

HLY78, a Lycorine (HY-N0288) derivative, is a potent activator of the Wnt/β-catenin signaling pathway. HLY78 targets the DIX domain of Axin and promotes the Axin-LRP6 (lipoprotein receptor-related protein 6) association, thus promoting LRP6 phosphorylation and Wnt signal transduction. HLY78 can be used for subarachnoid hemorrhage (SAH) research[1][2][3].

IC_{50} & Target

Wnt/β-catenin[1]

In Vitro

HLY78 inhibits apoptosis in tumor cells and embryonic cells caused by carbon ion radiation through activation of the Wnt/β-catenin pathway[2].

HLY78 (20 μM, 0-48 h) significantly increases the colony formation ability by 2.78-fold and 2.88-fold for HGC-27 and AGS cells compared with the controls[3].

HLY78 (20 μM, 0-48 h) elevates the migration ability of HGC-27 and AGS cells[3].

HLY78 significantly increases TNKS expression, which is ameliorated by Dihydroartemisinin (HY-N0176)[3].

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

In Vivo

HLY78 (0-1.8 mg/kg, Intranasal injection, once) attenuates neuronal apoptosis and improves neurological deficits through the LRP6/GSK3β/β-catenin signaling pathway after SAH (subarachnoid hemorrhage) in rats[2].

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

Animal Model:	Adult male Sprague-Dawley rats (280-310 g, n=9/group, SAH model)[2]
Dosage:	0, 0.2, 0.6, and 1.8 mg/kg
Administration:	Intranasal injection, once, at 1 h post-SAH (subarachnoid hemorrhage)
Result:	Significantly attenuated the short-term and long-term neurobehavioral deficits, as well as the neuronal apoptosis after SAH at 0.6 mg/kg. Successfully delivered into the brain via intranasal administration at 0.6 mg/kg and was sufficient to significantly increase the phosphorylation of LRP6. Reversed the changes of the Bcl-2, Bax, and cleaved caspase 3 levels.

CUSTOMER VALIDATION

- Cancer Gene Ther. 2022 Dec 9.
- Neurosci Bull. 2020 Oct;36(10):1171-1181.
- J Cancer. 2021; 12(24):7334-7348.
- Brain Res Bull. 2020 Sep;162:107-114.
- Oncol Lett. 2021 Oct;22(4):688.

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REFERENCES

[1]. Luo X, et al. HLY78 Attenuates Neuronal Apoptosis via the LRP6/GSK3β/β-Catenin Signaling Pathway After Subarachnoid Hemorrhage in Rats. Neurosci Bull. 2020 Oct;36(10):1171-1181.

[2]. Ma Y, et al. Dihydroartemisinin suppresses proliferation, migration, the Wnt/β-catenin pathway and EMT via TNKS in gastric cancer. Oncol Lett. 2021 Oct;22(4):688.

[3]. Wang S, et al. Small-molecule modulation of Wnt signaling via modulating the Axin-LRP5/6 interaction. Nat Chem Biol. 2013 Sep;9(9):579-85.

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