(S)-Oxiracetam

Cat. No.: HY-126049

CAS No.: 88929-35-5

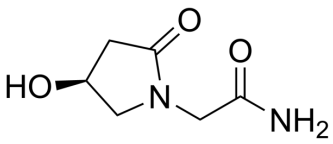
Molecular Formula: $C_6H_{10}N_2O_3$

Molecular Weight: 158.16

Target: Apoptosis

Pathway: Apoptosis

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (1580.68 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	6.3227 mL	31.6136 mL	63.2271 mL
	5 mM	1.2645 mL	6.3227 mL	12.6454 mL
	10 mM	0.6323 mL	3.1614 mL	6.3227 mL

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

BIOLOGICAL ACTIVITY

Description

(S)-oxiracetam (S-ORC) is an inhibitor targeting apoptosis. S-ORC reduces brain infarct size and lessens neurological dysfunction in middle cerebral artery occlusion/reperfusion (MCAO/R) models. S-ORC prevents neuronal apoptosis via activating PI3K/Akt/GSK3β signaling pathway via α7 nAChR after ischemic stroke. S-ORC can prevent neuronal death after ischemic stroke[1].

In Vitro

(S)-Oxiracetam (1, 10, 100 μM; 24 h) protects fetal rat primary cortical neurons against OGD/R injury and activates PI3K/Akt/GSK3β signal pathway was α7 nAChR dependent[1].

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

Cell Viability Assay[1]

Cell Line:	fetal rat primary cortical neurons
Concentration:	1, 10, 100 μM
Incubation Time:	24 h
Result:	Increased the survival of cortical neurons (58.82%, 64.82% and 79.15%, respectively)

	compared with the OGD/R group.

Cell Cytotoxicity Assay[1]

Cell Line:	fetal rat primary cortical neurons
Concentration:	1, 10, 100 μM
Incubation Time:	24 h
Result:	Decreased the LDH activity to 567.59 U/L, 484.89 U/L and 428.15 U/L at 1 μM, 10 μM and 100 μM, respectively, compared with the OGD/R group (725.22 U/L).

Apoptosis Analysis[1]

Cell Line:	fetal rat primary cortical neurons
Concentration:	1, 10, 100 μM
Incubation Time:	24 h
Result:	Reduced the apoptotic rate in cortical neurons dependent on α7 nAChR (34.48%, 16.15% and 10.05%, respectively) compared with the OGD/R group.

Western Blot Analysis[1]

Cell Line:	fetal rat primary cortical neurons
Concentration:	1, 10, 100 μM
Incubation Time:	24 h
Result:	Increased the expression of α7 nAChR (46.95%, 58.67% and 72.97%, respectively for the 1 μM, 10 μM and 100 μM) and the phosphorylation of PI3K, Akt and GSK3β dependent on α7 nAChR compared with the OGD/R group.

In Vivo

(S)-Oxiracetam (0.12, 0.24, 0.48 g/kg; i.v., once daily for 7 days) reduces the infarct size and lessens behavior dysfunction significantly, inhibits neuronal apoptosis, and activates PI3K/Akt/GSK3β signal pathway in Male Sprague-Dawley rats-induced MACO/R models[1].

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

Animal Model:	scopolamine-induced amnesia models in Swiss albino mice[1].
Dosage:	0.12, 0.24, 0.48 g/kg, once daily for 7 days
Administration:	Intravenous injection (i.v.)
Result:	Decreased the infarct size of rats to 26.04 ± 1.07%, 21.66 ± 2.27%, 12.26 ± 5.59% compared with the sham group at 0.12 g/kg, 0.24 g/kg, 0.48 g/kg, respectively. Increased neurological scores compared with the MCAO/R group. Protected neurons from apoptosis compared to sham group. Increased the α7 nAChR and the phosphorylation of PI3K, Akt and GSK3β expression compared with MCAO/R group. Increased of GSH-PX concentration (189.54 units, 193.07 units, and 203.98 untis, respectively).

REFERENCES

[1]. Fan W, et al. S-oxiracetam ameliorates ischemic stroke induced neuronal apoptosis through up-regulating α7 nAChR and PI3K / Akt / GSK3β signal pathway in rats. Neurochem Int. 2018 May;115:50-60.

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