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Kainic acid

Cat. No.: HY-N2309

CAS No.: 487-79-6

Molecular Formula: $C_{10}H_{15}NO_4$

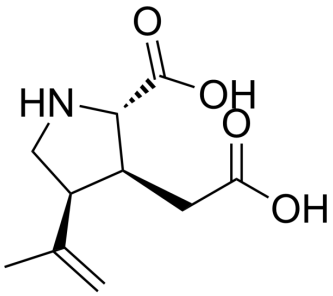
Molecular Weight: 213.23

Target: EAAT

Pathway: Membrane Transporter/Ion Channel

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 : 50 mg/mL (234.49 mM; ultrasonic and warming and heat to 60°C)

H_2O : 25 mg/mL (117.24 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.6898 mL	23.4489 mL	46.8977 mL
5 mM	0.9380 mL	4.6898 mL	9.3795 mL
10 mM	0.4690 mL	2.3449 mL	4.6898 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: ≥ 5 mg/mL (23.45 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 5 mg/mL (23.45 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 5 mg/mL (23.45 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Kainic acid is a potent excitotoxic agent. Kainic acid hydrate also is an agonist for a subtype of ionotropic glutamate receptor. Kainic acid induces seizures[1][2].

In Vivo

Kainic acid (5 mg/kg; i.p.; hourly at least 3 h until status epilepticus) induces seizures in rats[1].The kainic acid induced seizures model is a good tool to study temporal lobe epilepsy. The model can be reproduced in a variety of species through either systemic, intrahippocampal or intra-amygdaloid administrations. The systemic Kainic acid administration induced

model is similar with human temporal lobe epilepsy (TLE)[4][6].Kainic acid (5 nmoles, injections into the neostriatum, substantia nigra or cerebellum) shows that more than half of the compound disappeared from the injection site and the brain by 1/2 hour post injection, and less than radioactivity of 7 pmol/mg of tissue were found in other areas[3].

Induction of epilepsy model[5]

Background

Kainic acid, an analog of L-glutamate and an ionotropic KA receptor agonist, can damage hippocampal pyramidal neurons.

Specific Mmodeling Methods

Mice: C57BL/6J • male • 7 weeks old • 22 g body weight

Administration: 10 μg in 5 μL • i.c.v.

Note

(1) The right lateral brain ventricle is localized with a stereotactic instrument.

(2) After the operation, skin was sutured, and keep the mice under a warming place until they wake up.

(3) 48 hours after lateral ventricle injection, the mice are anaesthetized using Isoflurane and then sequentially intracardially perfused with saline and PFA (4%, 30 mL). Rapidly remove The mouse brain processed for paraffin embedding or frozen sections.

Modeling Indicators

Electroencephalogram (EEG) recording: Had higher local maximal amplitude and reduced spike frequency compared to the control group.

Histology analysis: Showed Triangulated pyknotic nuclei and cytoplasmic shrinkage in the hippocampal neuron, and induced neuronal loss.

□□□□:

□□□□:Sitagliptin (HY-13749)

Induction of epilepsy model[6]

Background

Kainic acid can bind to NMDA receptors which are abundant in neurons in the cornu ammonus of the hippocampus, amygdala, and pyriform cortex, and leading to variable levels of neuronal death in these regions.

Specific Mmodeling Methods

Rat: Sprague-Dawley rats • male

Administration: 10 mg/kg • i.p. • a single dose

Note

(1) Seizures occurres approximately 45 min after injection, typically lasted 2-3 hr.

(2) Kainic acid-treated animals may die during an acute period of intoxication, it's suggested to increase the number of animals per group.

(3) 72 hours later, the rats are anesthetized and immediately perfused transcardially with 50 mL 0.9% saline followed by 500 mL 0.1 m neutral phosphate-buffered formaldehyde (4%). And remove the brain and postfixed at 4°C overnight in the same fixative and dehydrated, embedded in paraffin, and cut into serial 6-μm-thick coronal sections at the level of the dorsal hippocampus.

Modeling Indicators

behavioral changes: Exhibited immobility and rigid postures firstly, followed by repetitive head nodding, ‘wet dog shakes’, and subsequent rearing and falling. Eventually, the rats developed generalized tonic–clonic seizures with continuous convulsions, lasting for several hours.

Histology analysis: Resulted in a loss of pyramidal neurons in fields CA1 and CA3 of the hippocampus.

Increaseed the size, arborization, and stainability of GFAP-immunoreactive cells.

□□□□:

□□□□:Melatonin (HY-B0075)

Animal Model:	8 weeks, 200-250 g male adult Wistar rats[1]
Dosage:	5 mg/kg
Administration:	I.p.; hourly at least 3 h until status epilepticus

Result:	Induced seizures in rats.

CUSTOMER VALIDATION

- Nat Neurosci. 2023 Apr;26(4):542-554.
- J Neuroinflammation. 2021 May 11;18(1):112.
- Biochem Biophys Res Commun. 2021 Feb 8;545:195-202.
- Brain Res. 12 August 2022, 148052.
- J Tradit Chin Med. 2022 Jun;42(3):379-388.

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[1]. Cincioğlu-Palabiyik M, et al. Chronic levetiracetam decreases hippocampal and testicular aromatase expression in normal but not kainic acid-induced experimental model of acute seizures in rats. Neuroreport. 2017 Sep 27;28(14):903-909.

[2]. Wang Q, et al. Kainic acid-mediated excitotoxicity as a model for neurodegeneration. Mol Neurobiol. 2005;31(1-3):3-16.

[3]. Scherer-Singler U, McGeer EG. Distribution and persistence of kainic acid in brain. Life Sci. 1979 Mar 12;24(11):1015-22.

[4]. Lévesque M, et al. The kainic acid model of temporal lobe epilepsy. Neurosci Biobehav Rev. 2013 Dec;37(10 Pt 2):2887-99.

[5]. Zheng Z, et al. The effect of dipeptidyl peptidase IV on disease-associated microglia phenotypic transformation in epilepsy. J Neuroinflammation. 2021 May 11;18(1):112.

[6]. Chung SY, et al. Melatonin attenuates kainic acid-induced hippocampal neurodegeneration and oxidative stress through microglial inhibition. J Pineal Res. 2003 Mar;34(2):95-102.

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