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Kynurenic acid sodium

Cat. No.: HY-107512

CAS No.: 2439-02-3

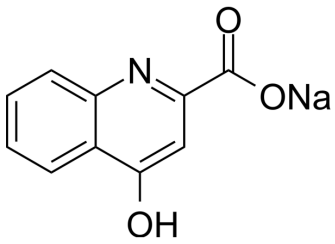
Molecular Formula: $C_{10}H_6NNaO_3$

Molecular Weight: 211.15

Target: iGluR; Apoptosis; Endogenous Metabolite; CXCR; GPR35

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling; Apoptosis; Metabolic Enzyme/Protease; GPCR/G Protein; Immunology/Inflammation

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 : 50 mg/mL (236.80 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	4.7360 mL	23.6798 mL	47.3597 mL
5 mM	0.9472 mL	4.7360 mL	9.4719 mL
10 mM	0.4736 mL	2.3680 mL	4.7360 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: ≥ 2.5 mg/mL (11.84 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (11.84 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (11.84 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Kynurenic acid sodium, an endogenous tryptophan metabolite, is a broad-spectrum antagonist targeting NMDA, glutamate, α7 nicotinic acetylcholine receptor. Kynurenic acid sodium is also an agonist of GPR35/CXCR8.

IC_{50} & Target		
	NMDA Receptor	Human Endogenous Metabolite

In Vitro

GPR35 functions as a receptor for the kynurenine pathway intermediate kynurenic acid. Kynurenic acid elicits calcium mobilization and inositol phosphate production in a GPR35-dependent manner in the presence of $G_{qi/o}$ chimeric G proteins.

	Kynurenic acid stimulates [^{35}S]guanosine 5′-O-(3-thiotriphosphate) binding in GPR35-expressing cells, an effect abolished by pertussis toxin treatment. Kynurenic acid also induces the internalization of GPR35[1]. KYNA's neuroinhibitory qualities and its neuroprotective and anticonvulsant effects are discovered using concentrations of the compound in the millimolar range. This, as well as the low affinity of KYNA at each of the three ionotropic glutamate receptors responsible for these effects [NMDA, alpha-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid (AMPA) and kainate], together with the realization that KYNA concentrations in the mammalian brain are in the sub-micromolar range, suggested that other receptors might serve as targets of endogenous Kynurenic acid. Kynurenic acid, with a shallower inhibition curve and non-competitively, antagonizes α7nAChRs on cultured hippocampal neurons with an IC_{50} in the low micromolar range[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Kynurenic acid affects the activity of leukocytes in the peripheral blood of mice, although the lowest one (2.5 mg/L) has the most profound influence in contrast to the highest one (250 mg/L), which produces the weakest effect. The lowest Kynurenic acid dose stimulates the proliferative response of T lymphocytes ($p<0.05$), after 7 and 28 days of administering the acid to the animals[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]	CHO-GPR35 stable cells are pretreated with or without pertussis toxin (100 ng/mL) for 16 h before harvesting. Cells are resuspended and homogenized in 10 mM Tris-HCl (pH 7.4), 1 mM EDTA followed by centrifugation at 1000 ×g for 10 min at 4 °C to remove nuclei and cellular debris. Membrane fractions are collected by spinning the supernatant at 38,000 ×g for 30 min and resuspended in 20 mM HEPES (pH 7.5) and 5 mM $MgCl_2$. 25 μg of membranes is incubated at room temperature for 1 h in assay buffer (20 mM HEPES, 5 m M$MgCl_2$, 0.1% bovine serum albumin (pH 7.5)) containing 3 μM GDP and 0.1 nM[^{35}S]GTPγS in the absence or presence of kynurenic acid. Reactions are terminated by vacuum filtration through GF/B filters, and the retained radioactivities are quantified on liquid scintillation counter[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Mouse: The experiment is performed on 160 male BALB/c mice, aged 10-12 weeks, with body weight of 22-26 g. The animals are maintained on a 12-h light/dark cycle at controlled temperature (20 ±1°C) and supplied with rodent chow and water ad libitum throughout the experiment. Mice are divided randomLy into four equal groups: control group (0) not receiving the Kynurenic acid, and three experimental groups administered the Kynurenic acid solution in drinking water at concentrations of 2.5, 25 or 250 mg/L. After 3, 7, 14 and 28 consecutive days of administration of the Kynurenic acid solution, 10 individuals from each group are sacrificed. The animals are anesthetized by inhalation of Aerrane and their blood is collected by heart puncture. Blood collected from five individuals of each group is used for the MTT assay, and from the next five for the flow cytometry[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Metab. 2023 Feb 13.
- J Anim Sci Biotechnol. 2023 Aug 5;14(1):111.
- In Vitro Cell Dev Biol Anim. 2023 Jun 8.

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REFERENCES

[1]. Wang J, et al. Kynurenic acid as a ligand for orphan G protein-coupled receptor GPR35. J Biol Chem. 2006 Aug 4;281(31):22021-8.

[2]. Albuquerque EX, et al. Kynurenic acid as an antagonist of α7 nicotinic acetylcholine receptors in the brain: facts and challenges. Biochem Pharmacol. 2013 Apr 15;85(8):1027-32.

[3]. Małaczewska J, et al. Effect of oral administration of kynurenic acid on the activity of the peripheral blood leukocytes in mice. Cent Eur J Immunol. 2014;39(1):6-13.

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