Riluzole hydrochloride

Cat. No.: HY-B0211A

CAS No.: 850608-87-6

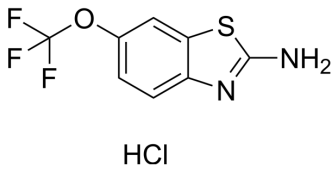
Molecular Formula: $C_8H_6ClF_3N_2OS$

Molecular Weight: 270.66

Target: Sodium Channel; GABA Receptor

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (369.47 mM; Need ultrasonic)
H_2O : 4.17 mg/mL (15.41 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.6947 mL	18.4734 mL	36.9467 mL
5 mM	0.7389 mL	3.6947 mL	7.3893 mL
10 mM	0.3695 mL	1.8473 mL	3.6947 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 (9.24 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (9.24 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.24 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Riluzole hydrochloride is an anticonvulsant agent and belongs to the family of use-dependent Na^+ channel blocker which can also inhibit GABA uptake with an IC_{50} of 43 μM.

IC_{50} & Target

Sodium channel[1]
IC50: 43 μM (GABA receptor)[1]

In Vitro

Riluzole hydrochloride is an anticonvulsant drug and belongs to the family of use-dependent Na^+ channel blocker which can also inhibit GABA uptake with an IC_{50} of 43 μM. At 20 μM, Riluzole hydrochloride inhibits peak autaptic IPSCs only slightly but

	prolongs IPSCs reliably. It is also found that Riluzole hydrochloride causes a strong, concentration-dependent, readily reversible enhancement of responses to 2 μM GABA. At higher concentrations of Riluzole hydrochloride, especially 300 μM, GABA currents exhibit apparent desensitization during prolonged co-exposure to 2 μM GABA and Riluzole hydrochloride. The EC_{50} of Riluzole hydrochloride potentiation of GABA responses is about 60 μM[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	In normal na ve rats, systemic injection of Riluzole hydrochloride (8 mg/kg, i.p.; n=6 rats) decreases the duration of ultrasonic but not audible vocalizations evoked by noxious stimulation of the knee joint compare to vehicle tested in the same rats (P<0.05). Systemic application of Riluzole hydrochloride (8 mg/kg, i.p.; n=19 rats) decreases the vocalizations of arthritic rats compare to predrug and vehicle significantly (P<0.05 to 0.001). Riluzole hydrochloride administered into the CeA significantly decreases the duration of audible and ultrasonic vocalizations evoked by noxious stimulation of the knee compare to predrug values (n=8 rats; P<0.05 to 0.01)[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Two-electrode voltage clamp of Xenopus oocytes expressing exogenous GABAA receptors is performed with a CA-1B high performance oocyte clamp. The extracellular recording solution is ND-96 medium. Riluzole hydrochloride is applied from a common tip via a gravity-driven multibarrel drug-delivery system. Data acquisition and analysis are performed with pCLAMP 6 software[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Adult male Sprague-Dawley rats (180 to 350 g) are housed in a temperature-controlled room and maintained on a 12-h day/night cycle with unrestricted access to food and water. Pain behaviors are measured before and 5 h after induction of a mono-arthritis in the left knee joint. To test the effects of systemic (intraperitoneal, i.p.) application of Riluzole hydrochloride, pain behaviors are measured 1 h postinjection of Riluzole hydrochloride in normal and arthritic animals. To determine effects of Riluzole hydrochloride into the amygdala, pain behaviors are measured 15 min after starting Riluzole hydrochloride application through a stereotaxically implanted microdialysis probe. To investigate site of action in the amygdala of systemically applied Riluzole hydrochloride, potassium channel blockers are administered into the amygdala 45 min after systemic application of Riluzole hydrochloride and pain behaviors are measured 15 min later, i.e., 1 h postinjection of riluzole (i.p.)[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Res. 2022 Apr 4.
- Nat Commun. 2023 Dec 12;14(1):8255.
- Proc Natl Acad Sci U S A. 2023 Oct 10;120(41):e2309773120.
- Free Radic Biol Med. 2024 Mar 24:S0891-5849(24)00141-2.
- Front Cell Neurosci. 2020 Oct 16;14:575626.

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REFERENCES

[1]. He Y, et al. Neuroprotective agent riluzole potentiates postsynaptic GABA(A) receptor function. Neuropharmacology. 2002 Feb;42(2):199-209.

[2]. Thompson JM, et al. Small-conductance calcium-activated potassium (SK) channels in the amygdala mediate pain-inhibiting effects of clinically available riluzole in a rat model of arthritis pain. Mol Pain. 2015 Aug 28;11:51.

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