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Lieferung & Zahlungsart

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Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
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Riluzole

Cat. No.: HY-B0211

CAS No.: 1744-22-5

Molecular Formula: $C_8H_5F_3N_2OS$

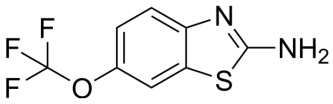
Molecular Weight: 234.2

Target: Sodium Channel; GABA Receptor

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

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 : 100 mg/mL (426.99 mM; Need ultrasonic)

H_2O : 1 mg/mL (4.27 mM; ultrasonic and adjust pH to 3 with HCl)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.2699 mL	21.3493 mL	42.6985 mL
5 mM	0.8540 mL	4.2699 mL	8.5397 mL
10 mM	0.4270 mL	2.1349 mL	4.2699 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 (10.67 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (10.67 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.67 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Riluzole 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 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 inhibits peak autaptic IPSCs only slightly but prolongs IPSCs reliably. It is also found that Riluzole causes a strong, concentration-dependent, readily reversible enhancement of responses to 2 μM GABA. At higher concentrations of Riluzole, especially 300 μM, GABA currents exhibit apparent desensitization during prolonged co-exposure to 2 μM GABA and Riluzole. The EC_{50} of Riluzole 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 (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 (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 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 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, pain behaviors are measured 1 h postinjection of Riluzole in normal and arthritic animals. To determine effects of Riluzole into the amygdala, pain behaviors are measured 15 min after starting Riluzole application through a stereotaxically implanted microdialysis probe. To investigate site of action in the amygdala of systemically applied Riluzole, potassium channel blockers are administered into the amygdala 45 min after systemic application of Riluzole 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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