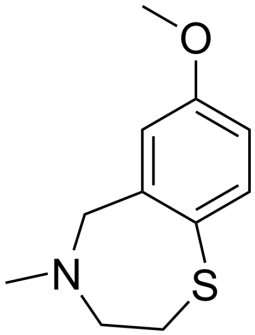
S107

Cat. No.:	HY-15292		
CAS No.:	927871-76-9		
Molecular Formula:	$C_{11}H_{15}NOS$		
Molecular Weight:	209.31		
Target:	Others		
Pathway:	Others		
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 (477.76 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	4.7776 mL	23.8880 mL	47.7760 mL
	5 mM	0.9555 mL	4.7776 mL	9.5552 mL
	10 mM	0.4778 mL	2.3888 mL	4.7776 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 (11.94 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (11.94 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (11.94 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

S107 is an orally available, blood brain barrier-permeable compound, which stabilizes RyR2 channels by enhancing the binding of calstabin 2 to the mutant Ryr2-R2474S channel. S107 inhibits Ca^{2+} leakage from the sarcoplasmic reticulum (SR) and prevents cardiac arrhythmias and raises the seizure threshold[1][2].

In Vitro

S107 is a small compound that enhances calstabin2 binding to RyR2 at low nanomolar concentrations and failed to interact with over 400 receptors, enzymes, and ion channels in screens using up to 10 μM of the compound[1].
S107 exerts an antiarrhythmic effect on CPVT-hiPSC-CMs. Pre-incubation with 10 μM S107, which stabilizes the closed state

	of the ryanodine receptor 2, significantly decreases the percentage of CPVT-hiPSC-CMs presenting DADs to 25%[2]. S107 increases FKBP12 binding to RyR1 in SR vesicles in the presence of reduced glutathione and the NO-donor NOC12, with no effect in the presence of oxidized glutathione. S107 can reverse the harmful effects of redox active species on SR Ca^{2+} release in skeletal muscle by binding to RyR1 low affinity sites[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	S107, which prevents a leak in the channel but does not block the channel or alter normal Ca^{2+} signaling, is able to inhibit both seizures and arrhythmias in the mutant mice[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [1]	Mice: To test for protection against seizures using S107, osmotic pumps are implanted, and mice are pretreated with S107 5 mg/kg/h for 1 week prior to seizure susceptibility testing. Phase 4 seizures associated with death could be avoided through intubation and artificial breathing, indicating diaphragm failure during sustained seizures as a potential cause of death. Mice are directly observed and videorecorded for later review and latency classification during a 60-minute observation period[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Commun. 2023 Feb 23;14(1):1020.

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REFERENCES

[1]. Lehnart SE, et al. Leaky Ca^{2+} release channel/ryanodine receptor 2 causes seizures and sudden cardiac death in mice. J Clin Invest. 2008 Jun;118(6):2230-45.

[2]. Sasaki K, et al. Patient-Specific Human Induced Pluripotent Stem Cell Model Assessed with Electrical Pacing Validates S107 as a Potential Therapeutic Agent for Catecholaminergic Polymorphic Ventricular Tachycardia. PLoS One. 2016 Oct 20;11(10):e0164795.

[3]. Mei Y, et al. Stabilization of the skeletal muscle ryanodine receptor ion channel-FKBP12 complex by the 1,4-benzothiazepine derivative S107. PLoS One. 2013;8(1):e54208.

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