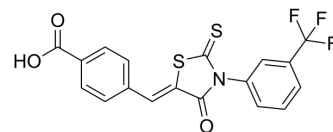


CFTR(inh)-172

Cat. No.:	HY-16671
CAS No.:	307510-92-5
Molecular Formula:	C ₁₈ H ₁₀ F ₃ NO ₃ S ₂
Molecular Weight:	409.4
Target:	CFTR; Autophagy
Pathway:	Membrane Transporter/Ion Channel; Autophagy
Storage:	<div>Powder</div> <div>-20°C 3 years</div> <div>4°C 2 years</div> <div>In solvent</div> <div>-80°C 6 months</div> <div>-20°C 1 month</div>



SOLVENT & SOLUBILITY

In Vitro	DMSO : 50 mg/mL (122.13 mM; Need ultrasonic)				
	Preparing Stock Solutions	<div>Solvent Concentration</div> <div>Mass</div>	1 mg	5 mg	10 mg
		1 mM	2.4426 mL	12.2130 mL	24.4260 mL
		5 mM	0.4885 mL	2.4426 mL	4.8852 mL
		10 mM	0.2443 mL	1.2213 mL	2.4426 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 (6.11 mM); Suspended solution; Need ultrasonic				
	2. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (6.11 mM); Clear solution				

BIOLOGICAL ACTIVITY

Description	CFTR(inh)-172 is a potent and selective blocker of the CFTR chloride channel; reversibly inhibits CFTR short-circuit current in less than 2 minutes with a K _i of 300 nM.
IC ₅₀ & Target	K _i : 300 nM (CFTR) ^[1]
In Vitro	Inhibition by CFTR(inh)-172 is complete in approximately 10 minutes (t _{1/2} =4 minutes) and is reversed after washout with t _{1/2} approximately 5 minutes. CFTRinh-172 is nontoxic to FRT cells after 24 hours at concentrations up to 100 μM ^[1] . CFTR(inh)-172 does not alter CFTR unitary conductance (8 pS), but reduces open probability by > 90% with K _i =0.6 μM. This effect is due to increased mean channel closed time without changing mean channel open time. The K _i values for inhibition of Cl ⁻ current in wild-type, G551D, and G1349D CFTR are about 0.5 μM; however, K _i is significantly reduced to 0.2 μM for vF508 CFTR ^[2] .

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

A single intraperitoneal injection of CFTR(inh)-172 (250 µg/kg) in mice reduces by more than 90% cholera toxin-induced fluid secretion in the small intestine over 6 hours. CFTR(inh)-172 is nontoxic at high concentrations in mouse models. CFTRinh-172 significantly reduces fluid secretion to that in saline control loops, whereas an inactive CFTRinh-172 analog does not inhibit fluid secretion^[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay ^[1]

CFTR(inh)-172 is diluted in DMSO as a 10 mM stock solution and diluted with appropriate medium. Fischer rat thyroid (FRT) cells coexpressing human wild-type CFTR and the halide indicator YFP-H148Q are generated. Cell toxicity is assayed by the dihydrorhodamine method at 24 hours after cell incubation with 0–1,000 µM inhibitor CFTR(inh)-172^[1].

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Animal Administration ^[1]

Mice: Animal toxicity is assessed by measurement of serum chemistries and hematology in mice at 5 days after daily intraperitoneal injections with 0-1,000 µg/kg CFTR(inh)-172^[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Stem Cell. 2019 Sep 5;25(3):373-387.e9.
- J Clin Invest. 2023 Nov 14:e171249.
- PLoS Biol. 2021 Feb 16;19(2):e3001090.
- Int J Mol Sci. 2022 Feb 23;23(5):2442.
- ACS Omega. 2023 Nov 25.

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REFERENCES

[1]. Ma T, et al. Thiazolidinone CFTR inhibitor identified by high-throughput screening blocks cholera toxin-induced intestinal fluid secretion. J Clin Invest. 2002 Dec;110(11):1651-8.

[2]. Taddei A, et al. Altered channel gating mechanism for CFTR inhibition by a high-affinity thiazolidinone blocker. FEBS Lett. 2004 Jan 30;558(1-3):52-6.

Caution: Product has not been fully validated for medical applications. For research use only.

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