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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
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CFTR(inh)-172

Cat. No.: HY-16671

CAS No.: 307510-92-5

Molecular Formula: $C_{18}H_{10}F_3NO_3S_2$

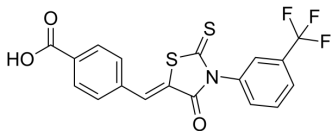
Molecular Weight: 409.4

Target: CFTR; Autophagy

Pathway: Membrane Transporter/Ion Channel; Autophagy

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (122.13 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	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

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

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