Ivacaftor

Cat. No.: HY-13017

CAS No.: 873054-44-5

Molecular Formula: $C_{24}H_{28}N_2O_3$

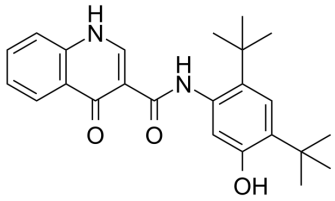
Molecular Weight: 392.49

Target: CFTR; Autophagy

Pathway: Membrane Transporter/Ion Channel; Autophagy

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (127.39 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5478 mL	12.7392 mL	25.4784 mL
	5 mM	0.5096 mL	2.5478 mL	5.0957 mL
	10 mM	0.2548 mL	1.2739 mL	2.5478 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 (6.37 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.37 mM); Clear solution
3. Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (6.37 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Ivacaftor (VX-770) is a potent and orally bioavailable CFTR potentiator, targeting G551D-CFTR and F508del-CFTR with EC_{50}s of 100 nM and 25 nM, respectively.

IC_{50} & Target

EC50: 100 nM (G551D-CFTR), 25 nM (F508del-CFTR)[1]

In Vitro

Ivacaftor (10 μM) increases the PC secretion activity by 3-fold for ABCB4-G535D, 13.7-fold for ABCB4-G536R, 6.7-fold for ABCB4-S1076C, 9.4-fold for ABCB4-S1176L, and 5.7-fold for ABCB4-G1178S. Ivacaftor corrects the functional defect of ABCB4

	mutants[1]. Ivacaftor (10 μM) significantly increases CFTR activity in W1282X-expressing cells compared to R1162X CFTR cells [2]. Ivacaftor shows no significant activity against 160 targets tested including the $GABA_A$ benzodiazepine receptor. Ivacaftor increases the chloride secretion with an EC_{50} of 0.236 ± 0.200 μM, a 10-fold shift in potency compared to the F508del HBEs[3]. In recombinant cells, VX-770 increases CFTR channel open probability (Po) in both the F508del processing mutation and the G551D gating mutation. VX-770 increases forskolin-stimulated I_T in temperature-corrected F508del-FRT cells by appr 6-fold with an EC_{50} of 25 nM[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Ivacaftor (1-200 mg/kg, p.o.) exhibits good oral bioavailability in rat[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Biomed Pharmacother. 2023 Aug 30;166:115399.
- Front Cell Dev Biol. 2021 May 11;9:678209.
- J Cell Sci. 2022 Jan 21;jcs.259002.
- Org Process Res Dev. 2019, 23, 11, 2302-2322.
- University of Kentucky. Master of Science in Medical Sciences. 2022 Aug.

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REFERENCES

[1]. Delaunay JL, et al. Functional defect of variants in the adenosine triphosphate-binding sites of ABCB4 and their rescue by the cystic fibrosis transmembrane conductance regulator potentiator, ivacaftor (VX-770). Hepatology. 2017 Feb;65(2):560-570

[2]. Mutyam V, et al. Therapeutic benefit observed with the CFTR potentiator, ivacaftor, in a CF patient homozygous for the W1282X CFTR nonsense mutation. J Cyst Fibros. 2017 Jan;16(1):24-29

[3]. Hadida S, et al. Discovery of N-(2,4-di-tert-butyl-5-hydroxyphenyl)-4-oxo-1,4-dihydroquinoline-3-carboxamide (VX-770, ivacaftor), a potent and orally bioavailable CFTR potentiator. J Med Chem. 2014 Dec 11;57(23):9776-9

[4]. Van Goor F, et al. Rescue of CF airway epithelial cell function in vitro by a CFTR potentiator, VX-770. Proc Natl Acad Sci U S A. 2009 Nov 3;106(44):18825-30.

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