KM11060

Cat. No.: HY-19970

CAS No.: 774549-97-2

Molecular Formula: $C_{19}H_{17}Cl_2N_3O_2S$

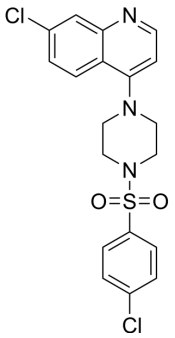
Molecular Weight: 422.33

Target: CFTR; Autophagy

Pathway: Membrane Transporter/Ion Channel; Autophagy

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 : 50 mg/mL (118.39 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3678 mL	11.8391 mL	23.6782 mL
	5 mM	0.4736 mL	2.3678 mL	4.7356 mL
	10 mM	0.2368 mL	1.1839 mL	2.3678 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 5 mg/mL (11.84 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

KM11060 is a corrector of the F508 deletion (F508del)-cystic fibrosis transmembrane conductance regulator (CFTR) trafficking defect. KM11060 can be used for the research of F508del-CFTR processing defect and development of cystic fibrosis research[1].

In Vitro

Small-molecule correctors such as KM11060 may serve as useful pharmacological tools in studies of the F508del-CFTR processing defect and in the development of cystic fibrosis therapeutics. KM11060 rescues F508del-CFTR trafficking in cultured cells and native epithelial tissues. KM11060 partially corrects F508del-CFTR processing and increases surface expression to 75% of that observed in cells incubated at low temperature. Up to 50% of the F508del-CFTR in cells treated with KM11060 was complex-glycosylated, indicating passage through the Golgi. KM11060 as a promising compound for further development of CF therapeutics. [1]

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

In Vivo	In LPS-induced acute lung inflammation, blockade of PSGL-1 (P-selectin glycoprotein ligand-1) or P-selectin, antagonism of PAF by WEB2086, or correction of mutated CFTR trafficking by KM11060 could significantly increase plasma lipoxin A4 levels in F508del relevant to wildtype mice. [2] MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Patent. US9987256B2.
- Patent. US20150328217A1.

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REFERENCES

[1]. Robert R, et al. Structural analog of sildenafil identified as a novel corrector of the F508del-CFTR trafficking defect. Mol Pharmacol. 2008 Feb;73(2):478-89.

[2]. Wu H, et al. Lipoxin A4 and platelet activating factor are involved in E. coli or LPS-induced lung inflammation in CFTR-deficient mice. PLoS One. 2014 Mar 26;9(3):e93003.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA