CMPD101

Cat. No.: HY-103045

CAS No.: 865608-11-3

Molecular Formula: $C_{24}H_{21}F_3N_6O$

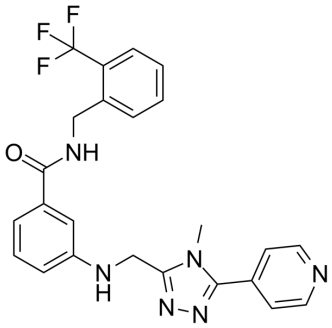
Molecular Weight: 466.46

Target: PKC; ROCK

Pathway: Epigenetics; TGF-beta/Smad; Cell Cycle/DNA Damage; Cytoskeleton; Stem Cell/Wnt

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 : 250 mg/mL (535.95 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.1438 mL	10.7190 mL	21.4381 mL
5 mM	0.4288 mL	2.1438 mL	4.2876 mL
10 mM	0.2144 mL	1.0719 mL	2.1438 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: ≥ 2.08 mg/mL (4.46 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.46 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.46 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CMPD101 is a potent, highly selective and membrane-permeable small-molecule inhibitor of GRK2/3 with IC_{50} of 18 nM and 5.4 nM, respectively. CMPD101 exhibits less selectively against GRK1, GRK5, ROCK-2 and PKCα with IC_{50}s of 3.1 μM , 2.3 μM, 1.4 μM and 8.1 μM, respectively. CMPD101 can be used for the study of heart failure[1].

IC_{50} & Target

GRK2	GRK3	PKCα	ROCK2
18 nM (IC_{50})	5.4 nM (IC_{50})	8.1 μM (IC_{50})	1.4 μM (IC_{50})

In Vitro

CMPD101 (100 μM; pre-20 mins) inhibit the internalization of β2AR, remarkably decreases the isoproterenol-induced formation of clathrin-coated vesicles and the β2AR-GFP fusion protein remained on the plasma membrane in HEK-B2 cell line[1].

CMPD101 (3-30 μM; pre-30 minutes) produced a robust phosphorylation of Ser375, which is partially inhibited by pretreatment of cells for 30 minutes with 3 μM Cmpd101 and fully blocked by pretreatment with 30 μM Cmpd101. It also inhibits phosphorylation of MOPr at Thr370, Thr376, and Thr379 residues[2].

CMPD101 (3-30 μM; pre-30 minutes) does not affect the DAMGO-induced increase in ERK1/2 and Elk-1 phosphorylation, at 30 μM, this compound produces a small increase in basal ERK1/2 phosphorylation in HEK 293 cells expressing HA-MOPrs[2].

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

Western Blot Analysis[2]

Cell Line:	HEK 293 cells stably expressing HA-tagged rat MOPr
Concentration:	3 μM, 30 μM
Incubation Time:	Pre-30 minutes
Result:	Surpressed Ser375 expression completely at a high dose.

Western Blot Analysis[2]

Cell Line:	HEK 293 cells stably expressing HA-tagged rat MOPr
Concentration:	3 μM, 30 μM
Incubation Time:	Pre-30 minutes
Result:	Had no effect on DAMGO-induced p-ERK1/2 and p-Elk-1 expression.

CUSTOMER VALIDATION

- J Exp Clin Cancer Res. 2020 Jun 9;39(1):107.
- Int J Mol Sci. 2022, 23(16), 8903.
- Biomolecules. 2022, 12(3), 426.

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REFERENCES

[1]. Okawa T, et al. Design, Synthesis, and Evaluation of the Highly Selective and Potent G-Protein-Coupled Receptor Kinase 2 (GRK2) Inhibitor for the Potential Treatment of Heart Failure. J Med Chem. 2017 Aug 24;60(16):6942-6990.

[2]. Yu Q, et al. Inhibition of prostatic smooth muscle contraction by the inhibitor of G protein-coupled receptor kinase 2/3, CMPD101.Eur J Pharmacol. 2018 Jul 15;831:9-19.

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

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