CID755673

Cat. No.: HY-12239

CAS No.: 521937-07-5

Molecular Formula: $C_{12}H_{11}NO_3$

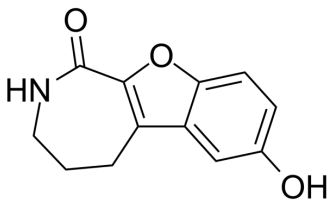
Molecular Weight: 217.22

Target: PKD

Pathway: Apoptosis

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 : 100 mg/mL (460.36 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.6036 mL	23.0181 mL	46.0363 mL
5 mM	0.9207 mL	4.6036 mL	9.2073 mL
10 mM	0.4604 mL	2.3018 mL	4.6036 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 (11.51 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (11.51 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (11.51 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CID755673 is a potent PKD inhibitor with IC_{50}s of 182 nM, 280 nM and 227 nM for PKD1, PKD2 and PKD3, respectively.

IC_{50} & Target

PKD1	PKD3	PKD2
182 nM (IC_{50})	227 nM (IC_{50})	280 nM (IC_{50})

In Vitro

CID755673 blocks phorbol ester-induced endogenous PKD1 activation in LNCaP cells in a concentration-dependent manner. CID755673 inhibits the known biological actions of PKD1 including phorbol ester-induced class IIa histone deacetylase 5

	nuclear exclusion, vesicular stomatitis virus glycoprotein transport from the Golgi to the plasma membrane, and the ilimaquinone-induced Golgi fragmentation. CID755673 inhibits prostate cancer cell proliferation, cell migration, and invasion[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Acute administration of the PKD inhibitor CID755673 to normal mice reduces both PKD1 and 2 phosphorylation in a time and dose-dependent manner. Chronic CID755673 administration to T2D db/db mice for two weeks reduces expression of the gene expression signature of PKD activation, enhances indices of both diastolic and systolic left ventricular function and is associated with reduced heart weight[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]	The radiometric kinase assay is carried out by coincubating 0.5 μCi of [γ-^{32}P]ATP, 20 μM ATP, 50 ng of purified recombinant human PKD (PKD1, PKD2, and PKD3) or CAMKIIα proteins, and 2.5 μg of Syntide-2 in 50 μL of kinase buffer that contains 50 mM Tris-HCl, pH 7.5, 4 mM $MgCl_2$, 10 mM β-mercaptoethanol. The reaction is carried out under conditions that the initial rate is within the linear kinetic range[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [1]	The wound-induced migration is triggered by scraping the cells with a plastic pipette tip, and the wound is imaged immediately. The DU145 cells are then are treated with or without CID755673 at different concentrations. The wound is imaged immediately (0 h) and at different intervals with an inverted phase-contrast microscope with a ×10 objective. At the end of the assay, cells are fixed with methanol and stained with crystal violet for a final image[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice: For acute inhibitor studies, C57BL6 mice are administered a single dose of vehicle (5% DMSO in PBS, pH 7.4), or the selective PKD inhibitor CID755673 at 1 or 10mg/kg body weight. Mice are killed one or four hr later and heart collected for later analysis. For chronic inhibitor experiments, 8-week old db/db mice receives vehicle or CID755673 at 1 or 10mg/kg bodyweight for 16 days, by daily intraperitoneal (i.p.) injection[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Brain Res Bull. 2020 Sep;162:141-150.
- Biochem Biophys Res Commun. 2022.
- Biochem Biophys Res Commun. 2020 Apr 2;524(2):280-287.
- Exp Ther Med. 2019 Apr;17(4):2511-2518.
- Int J Clin Exp Med. 2017;10(7):10528-10534.

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REFERENCES

[1]. Sharlow ER, et al. Potent and selective disruption of protein kinase D functionality by a benzoxoloazepinolone. J Biol Chem. 2008 Nov 28;283(48):33516-26.

[2]. Venardos K, et al. The PKD inhibitor CID755673 enhances cardiac function in diabetic db/db mice. PLoS One. 2015 Mar 23;10(3):e0120934.

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

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