CLK8

Cat. No.: HY-148765

CAS No.: 898920-65-5

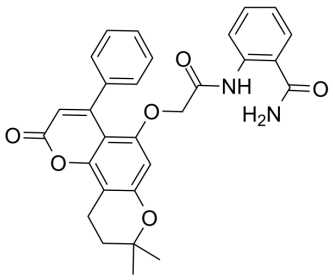
Molecular Formula: $C_{29}H_{26}N_2O_6$

Molecular Weight: 498.53

Target: Cryptochrome

Pathway: Metabolic Enzyme/Protease

Storage: 4°C, sealed storage, away from moisture and light
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (100.29 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0059 mL	10.0295 mL	20.0590 mL
	5 mM	0.4012 mL	2.0059 mL	4.0118 mL
	10 mM	0.2006 mL	1.0029 mL	2.0059 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 (5.01 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.01 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CLK8 is a potent and specific CLOCK inhibitor that can disrupt the interaction between CLOCK and BMAL1 and interfere with nuclear translocation of CLOCK. CLK8 can be used for the research of disorders associated with dampened circadian rhythms[1].

IC_{50} & Target

CLOCK[1]

In Vitro

CLK8 (10-40 μM; 4-6 d) enhances the amplitude of the Bmal1-dLuc signal in U2OS and NIH 3T3 cells in a dose-dependent manner with no period change[1].
CLK8 (10-40 μM) reduces BMAL1-CLOCK interaction, whereas the interaction between CLOCK-F80A, K220A and BMAL1 is not affected in HEK293T cells[1].
CLK8 (20 μM; 2 d) reduces nuclear localization of CLOCK in U2OS cells[1].

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

In Vivo

CLK8 (25 mg/kg; a single i.p.) decreases CLOCK levels in whole cell lysates of the mouse livers, whereas the levels of BMAL1 and CRY1 are unaltered[1].

CLK8 (5-1000 mg/kg; i.p.) exhibits no mortality or clinical signs (dyspnea, hyporeflexia, reduced locomotor activity, piloerection, hunched posture, and corneal opacity) at the doses of 5 and 25 mg/kg[1].

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

Animal Model:	Male C57BL/6J mice (8 weeks, 18-24 g)[1]
Dosage:	25 mg/kg
Administration:	A single i.p.
Result:	A decrease in CLOCK levels was detected in whole cell lysates of the mouse livers, whereas the levels of BMAL1 and CRY1 were unaltered. Decreased the abundance of CLOCK in the nucleus. The abundances of cytosolic and nuclear BMAL1 and CRY1 were unaltered. Decreased Cry1 transcriptional level.

REFERENCES

[1]. Doruk YU, et, al. A CLOCK-binding small molecule disrupts the interaction between CLOCK and BMAL1 and enhances circadian rhythm amplitude. J Biol Chem. 2020 Mar 13;295(11):3518-3531.

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