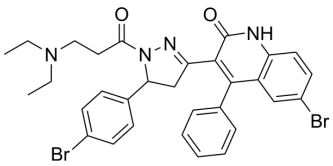
CCT020312

Cat. No.:	HY-119240
CAS No.:	324759-76-4
Molecular Formula:	$C_{31}H_{30}Br_2N_4O_2$
Molecular Weight:	650.4
Target:	PERK; Autophagy
Pathway:	Cell Cycle/DNA Damage; Autophagy
Storage:	Powder -20°C 3 years In solvent -80°C 1 year -20°C 6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (153.75 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.5375 mL	7.6876 mL	15.3752 mL
	5 mM	0.3075 mL	1.5375 mL	3.0750 mL
	10 mM	0.1538 mL	0.7688 mL	1.5375 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 (3.20 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.08 mg/mL (3.20 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (3.20 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CCT020312 is a selective EIF2AK3/PERK activator. CCT020312 elicits EIF2A phosphorylation in cells.

IC_{50} & Target

EIF2AK3/PERK[1][2].

In Vitro

Treatment of HT29 cells with CCT020312 for 24 hours reveals a concentration-dependent loss of P-S608-pRB signal, with a linear response between 1.8 and 6.1 μM[1].

CCT020312 treatment effectively inhibits cell proliferation (as measured at 96 hours) even if treatment is for 2 hours only with subsequent compound washout, indicating that CCT020312 is capable of eliciting durable rather than transient

cytostasis[1].
Treatment of HT29 cells with 10 μM CCT020312 for 24 hours reduces the amount of the G1/S cyclins D1, D2, E and A as well as the CDK catalytic subunit CDK2 and increased the level of the CDK inhibitor p27KIP1 present in such cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Treatment of 15-week-old wildtype mice with the PERK activator CCT020312 (1-5 mg/kg; i.p.; once daily for 3 days) leads to increased levels of phosphorylated PERK and NRF2 in brain homogenates[2].
P301S transgenic mice treated with CCT020312 (2 mg/kg; i.p.; once daily for 6 weeks) performes significantly better in Morris water maze[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	9-week-old P301S tau transgenic mice[2]
Dosage:	2 mg/kg
Administration:	Intraperitoneal injection; once daily for 6 weeks
Result:	P301S transgenic mice treated with CCT020312 performed significantly better in Morris water maze.

CUSTOMER VALIDATION

- Cell Metab. 2021 Mar 2;33(3):598-614.e7.
- Adv Sci (Weinh). 2023 May 11;e2205949.
- Cell Death Dis. 2020 Oct 13;11(10):847.
- J Transl Med. 2023 Feb 6;21(1):89.
- Cell Biol Toxicol. 2022 Jan 14.

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REFERENCES

[1]. Stockwell SR, et al.Mechanism-based screen for G1/S checkpoint activators identifies a selective activator of EIF2AK3/PERK signalling. PLoS One. 2012;7(1):e28568.

[2]. Bruch J, et al. PERK activation mitigates tau pathology in vitro and in vivo. EMBO Mol Med. 2017 Mar;9(3):371-384.

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