CMS-121

Cat. No.: HY-135981

CAS No.: 1353224-53-9

Molecular Formula: $C_{20}H_{19}NO_3$

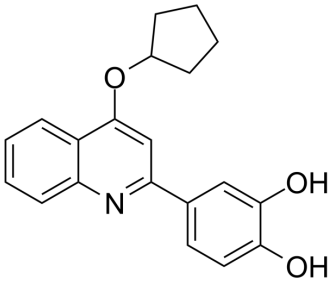
Molecular Weight: 321.37

Target: Acetyl-CoA Carboxylase

Pathway: Metabolic Enzyme/Protease

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 : 50 mg/mL (155.58 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.1117 mL	15.5584 mL	31.1168 mL
	5 mM	0.6223 mL	3.1117 mL	6.2234 mL
	10 mM	0.3112 mL	1.5558 mL	3.1117 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 (6.47 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (6.47 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CMS-121 is a quinolone derivative and an orally active acetyl-CoA carboxylase 1 (ACC1) inhibitor. CMS-121 protects HT22 cells against ischemia and oxidative damage with EC_{50} values of 7 nM and 200 nM, respectively. CMS-121 has strong neuroprotective, anti-inflammatory, antioxidative and renoprotective activities[1][2][3].

IC_{50} & Target

Acetyl-CoA carboxylase 1 (ACC1)[1]

In Vitro

CMS-121 (1 μM; 4 hours; HT22 cells) treatment increases the phosphorylation of ACC1 at serine 79. CMS-121 can increase acetyl-CoA in cells[1].

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

Western Blot Analysis[1]

Cell Line:	HT22 cells
Concentration:	1 μM
Incubation Time:	4 hours
Result:	Increases the phosphorylation of ACC1 at serine 79.

In Vivo

CMS-121 (~20 mg/kg; oral administration; daily; for 4 months; female SAMP8 mice) treatment reduces cognitive decline as well as metabolic and transcriptional markers of aging in the brain when administered to rapidly aging SAMP8 mice. CMS-121 preserves mitochondrial homeostasis by regulating acetyl-coenzyme A (acetyl-CoA) metabolism[1].

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

Animal Model:	Female SAMP8 mice (9 months old)[1]
Dosage:	~20 mg/kg/day
Administration:	Oral administration; daily; for 4 months
Result:	Reduced cognitive decline as well as metabolic and transcriptional markers of aging in the brain.

REFERENCES

[1]. Currais A, et al. Elevating acetyl-CoA levels reduces aspects of brain aging. Elife. 2019 Nov 19;8. pii: e47866.

[2]. Chiruta C, et al. Chemical modification of the multitarget neuroprotective compound fisetin. J Med Chem. 2012 Jan 12;55(1):378-89.

[3]. Prior M, et al. Back to the future with phenotypic screening. ACS Chem Neurosci. 2014 Jul 16;5(7):503-13.

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