CA77.1

Cat. No.: HY-134923

CAS No.: 2412270-22-3

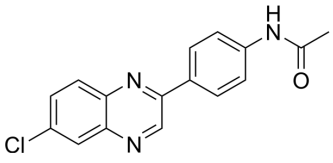
Molecular Formula: $C_{16}H_{12}ClN_3O$

Molecular Weight: 297.74

Target: Autophagy

Pathway: Autophagy

Storage: 4°C, protect from light
* In solvent : -80°C, 2 years; -20°C, 1 year (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 5 mg/mL (16.79 mM; ultrasonic and warming and heat to 60°C)
Ethanol : < 1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.3586 mL	16.7932 mL	33.5864 mL
5 mM	0.6717 mL	3.3586 mL	6.7173 mL
10 mM	0.3359 mL	1.6793 mL	3.3586 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 45% PEG300 >> 5% Tween-80 >> 50% saline
Solubility: 2 mg/mL (6.72 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

CA77.1 is a potent, brain-penetrant and orally active chaperone-mediated autophagy (CMA) activator with favorable pharmacokinetics. CA77.1 is a derivative of AR7 (HY-101106) and can increase the expression of the lysosomal receptor LAMP2A in lysosomes. CA77.1 improves behavior and neuropathology in PS19 mice model and can be used for alzheimer's disease research[1].

In Vitro

CA77.1 (0-30 μM; 16 hours) activates CMA in a dose-and time-dependent manner to NIH 3T3 cells stably expressing the KFERQ-PS-Dendra reporter. The CMA activity is quantified as the average of fluorescent puncta per cell[1].
CA77.1 (20 μM; 6 hours) does not alter on LC3-II expression, and does not effects autophagic flux in NIH 3T3 cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

CA77.1? (oral gavage; 10 mg/kg;single dose) demonstrates brain penetrance with favorable pharmacokinetics. The C_{max}, AUC_{last}, T_{max} and $T_{1/2}$ are 3534 ng/g, 8338 h*ng/g, 1 hour and 1.89 hour, respectively[1].
CA77.1 (oral gavage; 30 mg/kg; 6 months) normalizes the previously described locomotor hyperactivity of PS19 mice to

control levels. And it reduces the levels and number of neurons containing pathogenic tau conformations in the hippocampus, amygdala, and piriform cortex. And the higher number of microglial cells and presence of large Iba1-positive cells with rod-like dystrophic morphology in vehicle-treated PS19 mice are reduced upon CA77.1 treatment[1].

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

Animal Model:	9-month-old CTR or PS19 mice[1]
Dosage:	30 mg/kg
Administration:	Oral gavage; 30 mg/kg; 6 months
Result:	Improved behavior and neuropathology in a mouse model of frontotemporal-dementia-related proteotoxicity.

CUSTOMER VALIDATION

- Sci Adv. 2023 Oct 6;9(40):eadi8343.
- Cell Rep. 2023 Aug 16;42(8):112998.

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REFERENCES

[1]. Mathieu Bourdenx, et al. Chaperone-mediated autophagy prevents collapse of the neuronal metastable proteome.Cell. 2021 May 13;184(10):2696-2714.e25.

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