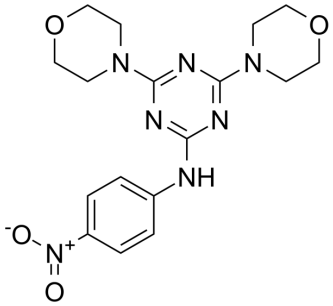
MHY1485

Cat. No.:	HY-B0795
CAS No.:	326914-06-1
Molecular Formula:	$C_{17}H_{21}N_7O_4$
Molecular Weight:	387.39
Target:	mTOR; Autophagy
Pathway:	PI3K/Akt/mTOR; Autophagy
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 : 7.69 mg/mL (19.85 mM; Need ultrasonic)
H_2O : 1 mg/mL (2.58 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5814 mL	12.9069 mL	25.8138 mL
	5 mM	0.5163 mL	2.5814 mL	5.1628 mL
	10 mM	0.2581 mL	1.2907 mL	2.5814 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: ≥ 0.77 mg/mL (1.99 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.77 mg/mL (1.99 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

MHY1485 is a potent cell-permeable mTOR activator that targets the ATP domain of mTOR. MHY1485 inhibits autophagy by suppression of fusion between autophagosomes and lysosomes[1].

IC_{50} & Target	mTORC1	mTORC2	Autophagy

In Vitro

MHY1485 (10 μM; 4 hours) shows that GCDC-induced autophagic activity is inhibited by upregulating p-mTOR expression and downregulating LC3 and p62 expression in HCC cells[1].
MHY1485 (5 μM; 6 hours) increases the LC3II/LC3I ratio in a dose and time-dependently manner due to presumably inhibited LC3II degradation in rat liver Ac2F cells[2].

MHY1485 (0.5-2 μM; 6 hours) increases the phosphorylation of mTOR at ser2448 and upregulates the level of phosphorylation of 4E-BP1 in a dose-dependently manner in Ac2F cells[2].

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

Western Blot Analysis[1]

Cell Line:	HCC cells
Concentration:	10 μM
Incubation Time:	4 hours
Result:	Upregulated p-mTOR and downregulated LC3 and p62 expression.

In Vivo

MHY1485 (intraperitoneal injection; 10 mg/kg, 2 days) blocks the autophagy signaling induced by follicle-stimulating hormone (FSH). It increases p-mTOR and p-S6K1 expression levels, whereas LC3 expression shows no marked change compared to that in the control group[3].

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

Animal Model:	4-week-old female ICR mice[3]
Dosage:	10 mg/kg, 2 days
Administration:	Intraperitoneal injection
Result:	Suppressed the autophagy level following FSH treatment.

CUSTOMER VALIDATION

- Cancer Lett. 2020 Jan 28;469:481-489.
- Cell Death Dis. 2022 Jan 12;13(1):53.
- Cell Death Dis. 2020 Mar 3;11(3):164.
- Cell Death Dis. 2019 Feb 13;10(2):140.
- Free Radic Biol Med. 2021 May 13;S0891-5849(21)00295-1.

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REFERENCES

[1]. Gao L, et al. Glycochenodeoxycholate promotes hepatocellular carcinoma invasion and migration by AMPK/mTOR dependent autophagy activation. Cancer Lett. 2019 Jul 10;454:215-223.

[2]. Choi YJ, et al. Inhibitory effect of mTOR activator MHY1485 on autophagy: suppression of lysosomal fusion. PLoS One. 2012;7(8):e43418.

[3]. Zhou J, et al.Administration of follicle-stimulating hormone induces autophagy via upregulation of HIF-1α in mouse granulosa cells.Cell Death Dis. 2017 Aug 17;8(8):e3001.

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

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