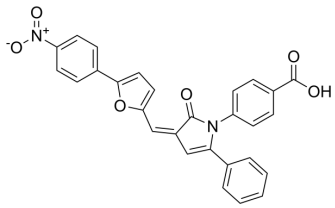
4E1RCat

Cat. No.:	HY-14427
CAS No.:	328998-25-0
Molecular Formula:	$C_{28}H_{18}N_2O_6$
Molecular Weight:	478.45
Target:	Eukaryotic Initiation Factor (eIF); Autophagy
Pathway:	Cell Cycle/DNA Damage; Autophagy
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 18.33 mg/mL (38.31 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0901 mL	10.4504 mL	20.9008 mL
	5 mM	0.4180 mL	2.0901 mL	4.1802 mL
	10 mM	0.2090 mL	1.0450 mL	2.0901 mL

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

BIOLOGICAL ACTIVITY

Description

4E1RCat is an inhibitor of cap-dependent translation, and inhibits eIF4E:eIF4GI interaction, with an IC_{50} an of ~4 μM.

IC_{50} & Target

eIF4

In Vitro

4E1RCat is an inhibitor of eIF4E:eIF4GI interaction, with an IC_{50} an of ~4 μM. 4E1RCat binding to eIF4E also interferes with eIF4G and 4E-BP binding. 4E1RCat inhibits ribosome recruitment to mRNA in a cap-dependent manner[1]. 4E1RCat blocks the capped mRNA translation, and the translation is activated by CDK1/CYCB1. Nearly all new protein synthesis in both mitosis and interphase is cap-dependent and -sensitive to 4E1RCat treatment, in HeLa and U2OS cells[2].

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

In Vivo

4E1RCat (15 mg/kg, i.p.) affacts chemosensitivity of $Pten^{+/-}$Eμ-Myc tumors in mice. 4E1RCat (15 mg/kg, i.p.) sensitizes $Pten^{+/-}$Eμ-Myc and $Tsc2^{+/-}$Eμ-Myc lymphomas to the cytotoxic effects of doxorubicin (Dxr), and 4E1RCat targets translation in mice [1].

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

PROTOCOL

Cell Assay [1]	$TSC2^{+/-}$Eμ-Myc and Eμ-Myc lymphomas are seeded in 96-well plates at 10^6 cells/mL in the presence of increasing concentrations of doxorubicin (Dxr) (ranging from 3.9 nM to 250 nM) and 4E1RCat (ranging from 78.13 nM to 10 000 nM) at a constant ratio of either 20:1 or 40:1. Twenty four hours later, a MTS assay is performed. To this end, Cell Proliferation Assay is added to the plates and the plates further incubated for up to 3 h, followed by measuring the OD_{490}. Values obtained are standardized against DMSO controls[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Mice[1] One million secondary $Pten^{+/-}$Eμ-Myc, $Tsc2^{+/-}$Eμ-Myc, or Eμ-Myc lymphoma cells are injected into the tail vein of 6-8 week old female C57BL/6 mice. When tumors are palpable, mice are treated with rapamycin (4 mg/kg daily for 5 d), 4E1RCat (15 mg/kg daily for 5 d), or doxorubicin (once at 10 mg/kg). Compounds are administered via intraperitoneal (i.p.) injection in 5.2% PEG 400/ 5.2% Tween 80. For combination studies, rapamycin or 4E1RCat are injected i.p. daily for five consecutive days, with doxorubicin being administered once on day two. Animals are palpated daily to monitor for the onset of tumors. Tumor-free survival is defined as the time between disappearance and reappearance of tumors. Data is analyzed using the log-rank test for statistical significance presented in Kaplan-Meier format[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Front Oncol. 2020 Jun 19;10:834.
- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.

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REFERENCES

[1]. Cencic R, et al. Reversing chemoresistance by small molecule inhibition of the translation initiation complex eIF4F. Proc Natl Acad Sci U S A. 2011 Jan 18;108(3):1046-51.

[2]. Shuda M, et al. CDK1 substitutes for mTOR kinase to activate mitotic cap-dependent protein translation. Proc Natl Acad Sci U S A. 2015 May 12;112(19):5875-82.

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