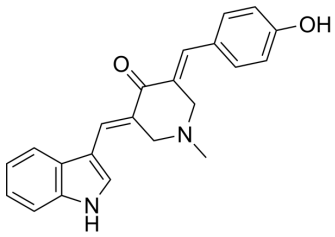
CUR5g

Cat. No.:	HY-152100
CAS No.:	1370032-20-4
Molecular Formula:	$C_{22}H_{20}N_2O_2$
Molecular Weight:	344.41
Target:	Autophagy
Pathway:	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 : 41.67 mg/mL (120.99 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.9035 mL	14.5176 mL	29.0352 mL
	5 mM	0.5807 mL	2.9035 mL	5.8070 mL
	10 mM	0.2904 mL	1.4518 mL	2.9035 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: ≥ 2.5 mg/mL (7.26 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CUR5g is a potent autophagy inhibitor. CUR5g selectively inhibits autophagosome degradation in cancer cells by blocking autophagosome-lysosome fusion. CUR5g blocks the recruitment of STX17 to autophagosomes via a UVRAG-dependent mechanism, resulting in the inability of autophagosomes to fuse with lysosomes. CUR5g improves the anticancer effect of Cisplatin (HY-17394) against A549 cells both in vitro and in vivo[1].

In Vitro

CUR5g (0-40 μM, 0-24 h) selectively induces autophagosome accumulation in cancer cells[1].
CUR5g (0-40 μM, 0-24 h) up-regulates LC3B-II and sequestosome 1 (SQSTM1) levels[1].
CUR5g (0-40 μM, 24 h) inhibits proliferation and migration of A549 cells, but dose not induce apoptosis or necrosis[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Autophagy Assay[1]

Cell Line:	A549 cells
Concentration:	0, 1, 5, 10, 20, and 40 μM
Incubation Time:	3, 6, 12, and 24 h
Result:	Induced extensive cytoplasmic vacuolization, and GFP-LC3B signal shifted from diffuse cytosolic staining to a punctate pattern outlining autophagosomes.

Western Blot Analysis[1]

Cell Line:	A549 cells
Concentration:	0, 1, 5, 10, 20, and 40 μM
Incubation Time:	0, 1, 3, 6, 12, and 24 h
Result:	Up-regulated LC3B-II and sequestosome 1 (SQSTM1) levels time- and dose-dependently. This increase was not the result of enhanced transcription, as mRNA expression of SQSTM1 and LC3B were not increased within CUR5g-exposed cells, suggesting that CUR5g might block autophagic flux rather than increase autophagosome formation.

Cell Proliferation Assay[1]

Cell Line:	A549 cells
Concentration:	0, 1, 5, 10, 20, and 40 μM
Incubation Time:	24 h
Result:	Exhibited great toxicity to A549 cells at 20 μM. Slightly decreased A549 cell number at 10 μM, while decreased the number of A549 cells significantly at 20 μM. Showed no discernable activity in healthy human umbilical vein endothelial cell (HUVEC) viability at 40 μM.

In Vivo

CUR5g (40 mg/kg, Injected via caudal vein, once every 2 days for up to 15 days) exhibits synergistic anticancer effects with Cisplatin (HY-17394) (1 mg/kg) and inhibits autophagic flux in vivo[1].

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

Animal Model:	BALB/c nude mice (4-week-old, A549 cells were subcutaneously injected into the right scapula of each nude mouse)[1]
Dosage:	40 mg/kg, CUR5g (40 mg/kg) and Cisplatin (1 mg/kg)
Administration:	Injected via caudal vein, once every 2 days for up to 15 days
Result:	Retarded the growth of xenografted tumors, whereas the combination treatment with Cisplatin almost completely inhibited tumor growth. Promoted the cisplatin sensitivity of A549 cells by inhibiting autophagic flux.

REFERENCES

[1]. Chen J, et al. CUR5g, a novel autophagy inhibitor, exhibits potent synergistic anticancer effects with cisplatin against non-small-cell lung cancer. Cell Death Discov. 2022 Oct 31;8(1):435.

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

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