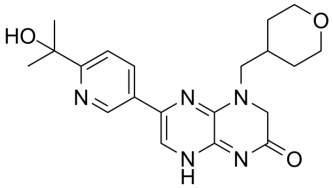
CC214-2

Cat. No.:	HY-145931
CAS No.:	1228012-18-7
Molecular Formula:	$C_{20}H_{25}N_5O_3$
Molecular Weight:	383.44
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 : 12.5 mg/mL (32.60 mM; ultrasonic and warming and heat to 80°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6080 mL	13.0399 mL	26.0797 mL
	5 mM	0.5216 mL	2.6080 mL	5.2159 mL
	10 mM	0.2608 mL	1.3040 mL	2.6080 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: ≥ 1.25 mg/mL (3.26 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.25 mg/mL (3.26 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CC214-2 is an oral active and selective mTOR kinase inhibitor. CC214-2 targets to both of mTORC1 (pS6) and mTORC2 (pAktS473). CC214-2 induces autophagy, which is a potential target for host-directed therapy (HDT) in tuberculosis. CC214-2 exhibits synergistic bactericidal and sterilizing activity agasinst tuberculosis (TB), and shortens the treatment duration. CC214-2 also inhibits Rapamycin (HY-10219)-resistant signaling and the growth of glioblastomas in vitro and in vivo[1][2].

IC_{50} & Target	mTORC1	mTORC2

In Vivo

CC214-2 (25 mg/kg, 50 mg/kg; po; once daily for 21 days) results tumor volume reductions in PC3 tumor xenograft model[1]. CC214-2 (30 mg/kg, 100 mg/kg; po; single dose) inhibits both mTORC1 (pS6) and mTORC2 (pAktS473) in vivo for at least 8 and 24 h, respectively[1].

CC214-2 (30 mg/kg; po) reduces M. tuberculosis CFU numbers and prevents mortality in mice with Chronic M. tuberculosis infection[1].
CC214-2 (50 mg/kg; po; once daily for 6 days) significantly reduces the growth of mouse U87EGFRvIII flank xenografts[2].
CC214-2 (100 mg/kg; po; once every 2 days for 6 days) inhibits mTORC1 and mTORC2 signaling in an intracranial glioblastoma model[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	U87EGFRvIII flank xenografts in mice[2]
Dosage:	50 mg/kg
Administration:	PO; once daily for 6 days
Result:	Inhibited tumor growth. Similarly activated autophagy in U87EGFRvIII xenografts.

REFERENCES

[1]. Gini B, et al. The mTOR kinase inhibitors, CC214-1 and CC214-2, preferentially block the growth of EGFRvIII-activated glioblastomas. Clin Cancer Res. 2013 Oct 15;19(20):5722-32.

[2]. Tasneen R, et al. Dual mTORC1/mTORC2 Inhibition as a Host-Directed Therapeutic Target in Pathologically Distinct Mouse Models of Tuberculosis. Antimicrob Agents Chemother. 2021;65(7):e0025321.

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