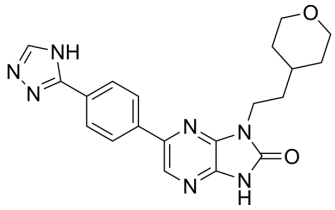
CC214-1

Cat. No.:	HY-154910
CAS No.:	1021920-32-0
Molecular Formula:	$C_{20}H_{21}N_7O_2$
Molecular Weight:	391.43
Target:	mTOR
Pathway:	PI3K/Akt/mTOR
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	CC214-1 is a potentially efficacious mTOR inhibitor that induces autophagy [1],with an IC_{50} is 0.002 μM. CC214-1 proved to be useful as an in vitro tool compound for the exploration of mTOR kinase biology. CC214-1 can be used for Glioblastoma study [2].
IC_{50} & Target	mTOR 0.002 μM (IC_{50})

In Vitro

CC214-1 (0, 0.1, 1, 2, 5, 10 μM, 8 h; 2 μM, 24 h) synergize with rapamycin (HY-10219), inhibiting mTORC1 signaling and tumor cell proliferation[1].

CC214-1 (2 μM, 24 h) -mediated sensitivity to growth arrest in glioblastoma cells due to EGFRvIII expression and loss of PTEN [1].

CC214-1 (5 μM, 0-48 h) massively lipidates LC3B-I to LC3B-II subtype and induces autophagy in GBM39 cells[1].

CC214-1 has an IC_{50} of mTOR is 0.002 μM[2].

CC214-1 (0-10 μM, 4days) is efficient in inhibiting T cell activation and the expression of T-cell activation markers[3].

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

Western Blot Analysis[1]

Cell Line:	glioblastoma
Concentration:	0, 0.1, 1, 2, 5, 10 μM; 2 μM
Incubation Time:	8 h; 24 h
Result:	Inhibited mTORC1 signaling in all glioblastoma cell lines tested, potently suppressing rapamycin-resistant 4E-BP1 and mTORC2 signaling. Inhibited mTORC1-dependent 4E-BP1 and S6 phosphorylation in EGFRvIII-expressing glioblastoma cells, as well as blocked glioblastoma cells overexpressing wild-type EGFR.

Immunofluorescence[1]

Cell Line:	glioblastoma
Concentration:	2 μM, 5 μM

Incubation Time:	4 h; 0, 4, 12, 24, 48 h
Result:	Induced a transient expression of LC3B-II isoform and the conjugation of Atg12 to Atg5 indicative of the stimulation of the autophagy flux in U87EGFRvIII cell line. Massively lipidated LC3B-I to LC3B-II subtype and induces autophagy in GBM39 cells.

Cell Cycle Analysis[3]

Cell Line:	glioblastoma
Concentration:	0-10 μM
Incubation Time:	4 day
Result:	Induced TEE cells in CD4+ and CD8+ T cell subsets.

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]. Mortensen DS, et al. Use of core modification in the discovery of CC214-2, an orally available, selective inhibitor of mTOR kinase. Bioorg Med Chem Lett. 2013 Mar 15;23(6):1588-91.

[3]. Herrero-Sánchez MC, et al. Effect of mTORC1/mTORC2 inhibition on T cell function: potential role in graft-versus-host disease control. Br J Haematol. 2016 Jun;173(5):754-68.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA