CPI-455

Cat. No.: HY-100421

CAS No.: 1628208-23-0

Molecular Formula: $C_{16}H_{14}N_4O$

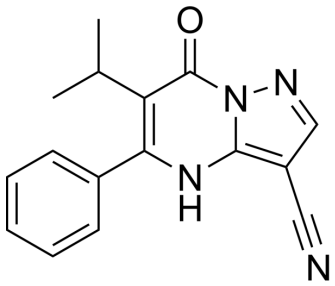
Molecular Weight: 278.31

Target: Histone Demethylase

Pathway: Epigenetics

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 : ≥ 34 mg/mL (122.17 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.5931 mL	17.9656 mL	35.9312 mL
5 mM	0.7186 mL	3.5931 mL	7.1862 mL
10 mM	0.3593 mL	1.7966 mL	3.5931 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 (8.98 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CPI-455 is a specific, pan-KDM5 inhibitor with an IC_{50} of 10 nM for KDM5A. CPI-455 mediates KDM5 inhibition, elevates global levels of H3K4me3, and decreases the number of drug-tolerant persister cancer cells in multiple cancer cell line models treated with standard chemotherapy or targeted agents[1].

IC_{50} & Target

KDM5

In Vitro

CPI-455 mediates KDM5 inhibition, elevates global levels of H3K4 trimethylation (H3K4me3) and decreases the number of DTPs in multiple cancer cell line models treated with standard chemotherapy or targeted agents[1].
CPI-455, with high measured affinity for the target KDM5 proteins. Within 24 hours, increases in H3K4me3, are observed after exposure to either of the two active compounds, CPI-455 and CPI-766, in a dosedependent manner. IC_{50} calculation for KDM5 Inhibitor CPI0455 in 3 luminal breast cancer cell lines MCF-7, T-47 and EFM-19 are 35.4, 26.19 and 16.13 μM,

	respectively[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Dual blockade of B7-H4 and KDM5B (CPI-455, 50/70 mg/kg, ip, daily) in mice elicits protective immunity[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Six-week-old male C57BL/6 mice (One- to 2-mm fragments of P. gingivalis–positive PDXs were implanted subcutaneously into the flank region of humanized mice.)
Dosage:	50 mg/kg or 70 mg/kg (combined with anti–B7-H4).
Administration:	IP, daily, 14-28 days.
Result:	Histopathology analysis revealed no inflammation in either group at 2 weeks in response to the primary infection. However, at 8 weeks after inoculation, mice receiving monotherapy exhibited mild inflammation, whereas the combined treatment presented with heavy to severe inflammation, which persisted at 12 and 16 weeks after challenge. Treatment with CPI-455 to selectively target H3K4-specific JmjC demethylases increased CXCL11, CXCL9, and CXCL10 following infection, with maximum levels observed 48 hours after infection.

CUSTOMER VALIDATION

- Oncogene. 2021 Apr;40(15):2711-2724.
- Blood Adv. 2021 Sep 14;5(17):3241-3253.
- Cancers. 2019 Jan 15;11(1):92.
- Inflammation. 2021 Apr 29.

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REFERENCES

[1]. Vinogradova M, et al. An inhibitor of KDM5 demethylases reduces survival of drug-tolerant cancer cells. Nat Chem Biol. 2016 Jul;12(7):531-8.

[2]. Benjamin R. Leadem. NOVEL HISTONE DEMETHYLASE INHIBITORS SYNERGISTICALLY

[3]. Xiang Yuan, et al. Blockade of Immune-Checkpoint B7-H4 and Lysine Demethylase 5B in Esophageal Squamous Cell Carcinoma Confers Protective Immunity against P. gingivalis Infection. Cancer Immunol Res. 2019 Sep;7(9):1440-1456.

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