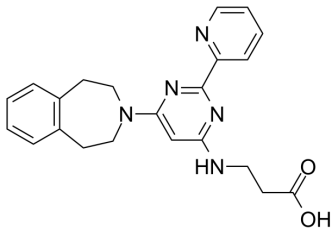
GSK-J1

Cat. No.:	HY-15648
CAS No.:	1373422-53-7
Molecular Formula:	$C_{22}H_{23}N_5O_2$
Molecular Weight:	389.45
Target:	Histone Demethylase
Pathway:	Epigenetics
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 : 50 mg/mL (128.39 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5677 mL	12.8386 mL	25.6772 mL
	5 mM	0.5135 mL	2.5677 mL	5.1354 mL
	10 mM	0.2568 mL	1.2839 mL	2.5677 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 (6.42 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.42 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

GSK-J1 is a potent inhibitor of H3K27me3/me2-demethylases JMJD3/KDM6B and UTX/KDM6A, with IC_{50} of 60 nM towards KDM6B.

IC_{50} & Target

IC50: 60 nM (KDM6B)[2]

In Vitro

GSK-J1 is selective for H3K27 demethylases of the KDM6 subfamily and specifically binds to endogenous JMJD3. GSK-J1 inhibits TNF-α production by human primary macrophages in an H3K27-dependent manner[1]. GSK-J1 inhibits the demethylase activity of KDM5C with 8.5-fold increased potency compared with that of KDM5B at 1 mM α-ketoglutarate, with IC_{50} of 11 μM and 94 μM, respectively[3].

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

PROTOCOL

Kinase Assay [1]

Purified JmjD3 (1 μM) and UTX (3 μM) is incubated with 10 μM peptide [BiotinKAPRKQLATKAARK(me3)SAPATGG] in 50 mM HEPES pH 7.5, 150 mM KCl, 50 μM $(NH_4)_2SO_4 \cdot FeSO_4 \cdot H_2O$, 1 mM 2-oxoglutarate, and 2 mM ascorbate (JmjD3, 3 minutes at 25°C; UTX, 20 minutes at 25°C) with various concentration of the inhibitor (0, 0.005, 0.01, 0.02, 0.05, 0.1 μM). 10 mM EDTA is added to stop the reaction. The reaction is desalted by zip tip and spotted on a MALDI plate with α-cyano-4-hydroxycinnamic acid MALDI matrix. Samples are analysed on a MALDI-TOF R system.

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

CUSTOMER VALIDATION

- Oncogene. 2021 Mar 12.
- Acta Pharmacol Sin. 2021 Apr 13.
- Front Mol Neurosci. 2017 Mar 13;10:51.
- J Chromatogr A. 2020 Feb 22;1613:460625.
- SSRN. 2021 Dec.

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REFERENCES

[1]. Kruidenier L, et al. A selective jumonji H3K27 demethylase inhibitor modulates the proinflammatory macrophage response. Nature. 2012 Aug 16;488(7411):404-8.

[2]. Heinemann B, et al. Inhibition of demethylases by GSK-J1/J4. Nature. 2014 Oct 2;514(7520):E1-2.

[3]. Horton JR, et al. Characterization of a Linked Jumonji Domain of the KDM5/JARID1 Family of Histone H3 Lysine 4 Demethylases. J Biol Chem. 2016 Feb 5;291(6):2631-46.

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