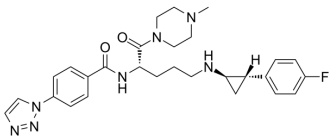
Bomedemstat

Cat. No.:	HY-109169
CAS No.:	1990504-34-1
Molecular Formula:	$C_{28}H_{34}FN_7O_2$
Molecular Weight:	519.61
Target:	Histone Demethylase; Apoptosis
Pathway:	Epigenetics; Apoptosis
Storage:	Pure form -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (192.45 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.9245 mL	9.6226 mL	19.2452 mL
	5 mM	0.3849 mL	1.9245 mL	3.8490 mL
	10 mM	0.1925 mL	0.9623 mL	1.9245 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

Bomedemstat (IMG-7289) is an orally active and irreversible lysine-specific demethylase 1 (LSD1) inhibitor. Bomedemstat can increase H3K4 and H3K9 methylation, and then alter gene expression. Bomedemstat shows anti-cancer activities, inhibits cancer cell proliferation and induces apoptosis[1][2].

In Vitro

Bomedemstat selectively inhibits proliferation and induces apoptosis of $Jak2^{V617F}$ cells by concomitantly increasing expression and methylation of p53[1].

Bomedemstat (50 nM-1 μM; 96 h) enhances survival, induces apoptosis via BCL-XL and PUMA in a TP53-dependent manner, and leads to cell cycle arrest[1].

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

Apoptosis Analysis[1]

Cell Line:	SET-2 cells
Concentration:	50 nM, 100 nM, and 1 μM
Incubation Time:	96 hours

Result:	Decreased levels of the antiapoptotic protein BCL-XL and increased levels of the pro-apoptotic protein PUMA.

In Vivo

Bomedemstat treatment (oral gavage; 45 mg/kg; once daily; 56 d) normalizes or improves blood cell counts, reduces spleen volumes, restores normal splenic architecture, and reduces bone marrow fibrosis[1].

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

Animal Model:	Mx-Jak2^{V617F} mice[1]
Dosage:	45 mg/kg
Administration:	Oral gavage; 45 mg/kg; once daily; 56 days
Result:	Reduced splenomegaly significantly with a few treated mice normalizing their spleen weight, the 56-day course led to partial restoration of lymph follicles and spleen architecture by histological examination.

REFERENCES

[1]. Yuan Fang, et al. LSD1/KDM1A inhibitors in clinical trials: advances and prospects. J Hematol Oncol. 2019 Dec 4;12(1):129.

[2]. Jonas S Jutzi,et al. LSD1 Inhibition Prolongs Survival in Mouse Models of MPN by Selectively Targeting the Disease Clone. Hemasphere.2018 Jun 8;2(3):e54.

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