Bomedemstat dihydrochloride

Cat. No.: HY-109169C

Molecular Formula: $C_{28}H_{36}Cl_2FN_7O_2$

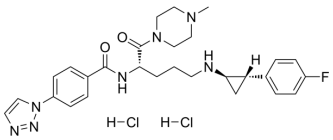
Molecular Weight: 592.54

Target: Histone Demethylase; Apoptosis

Pathway: Epigenetics; Apoptosis

Storage: -20°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (421.91 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.6876 mL	8.4382 mL	16.8765 mL
	5 mM	0.3375 mL	1.6876 mL	3.3753 mL
	10 mM	0.1688 mL	0.8438 mL	1.6876 mL

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

BIOLOGICAL ACTIVITY

Description

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

IC_{50} & Target

KDM1/LSD1

In Vitro

Bomedemstat dihydrochloride 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; SET-2 cells) dihydrochloride 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 (oral gavage; 45 mg/kg; once daily; 56 d) dihydrochloride 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]. 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.

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

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