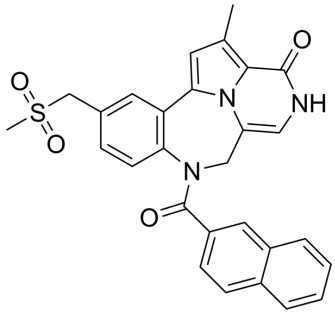
BET-IN-13

Cat. No.:	HY-152213
CAS No.:	2506823-08-9
Molecular Formula:	$C_{28}H_{23}N_3O_4S$
Molecular Weight:	497.56
Target:	Epigenetic Reader Domain
Pathway:	Epigenetics
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

BET-IN-13 is a potent BET inhibitor with an IC_{50} value of 1.6 nM. BET-IN-13 reduces LPS-induced TNF-α, IL-1β, IL-6, and NOS2 mRNA expression levels. BET-IN-13 shows anti-inflammatory activity. BET-IN-13 has the potential for the research of acute liver injury[1].

IC_{50} & Target

BRD4 BD1 57.4 nM (IC_{50})	BRD4 BD2 44.4 nM (IC_{50})	BRD2 BD1 79.3 nM (IC_{50})	BRD2 BD2 27.5 nM (IC_{50})
BRD3 BD1 45.6 nM (IC_{50})	BRD3 BD2 18.9 nM (IC_{50})	BRDT BD1 87.0 nM (IC_{50})	BRDT BD2 43.4 nM (IC_{50})

In Vitro

BET-IN-13 (compound 28) (1.1, 3.3, 10 μM, 2+6 h) reduces LPS (500 ng/ml) induced TNF-α, IL-1β, IL-6 and NOS2 mRNA expression levels in RAW264.7 cells[1].

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

RT-PCR[1]

Cell Line:	RAW264.7 cells
Concentration:	1.1, 3.3, 10 μM
Incubation Time:	Pre-treated for 2 h before stimulating with LPS for 6 h
Result:	Significantly reduced LPS (500 ng/ml) induced TNF-α, IL-1β, IL-6 and NOS2 mRNA expression levels in a dose-dependent manner.

In Vivo

BET-IN-13 (3 mg/kg; i.v.; once) shows good pharmacokinetic (PK) properties with a $T_{1/2}$ of 0.69 h, $AUC_{INF-obs}$ of 609 h*ng/mL and V_{ss} of 1717 mL/kg in mouse[1].

BET-IN-13 (37.5, 75 mg/kg; i.p.; once) reduces the inflammation and hepatic damage without obvious toxicity in PS/D-GalN(d-gaiactosamine)-induced acute liver failure (ALF) mouse[1].

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

Animal Model:	20-22g, Female C57BL/6J mice (LPS/D-GalN(d-gaiactosamine)-induced acute liver failure (ALF))[1]

Dosage:	37.5, 75 mg/kg
Administration:	I.p.; once
Result:	Reduced inflammatory responses associated with LPS/GalN-induced acute liver failure with the survival rate increased significantly to 69.2% for37.5 mg/kg and to 84.6% for 75 mg/kg.

REFERENCES

[1]. Chen C, et al. Cyclization strategy leads to highly potent Bromodomain and extra-terminal (BET) Bromodomain inhibitors for the treatment of acute liver injury. Eur J Med Chem. 2022 Dec 16;247:115023.

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