BRD3308

Cat. No.: HY-19618

CAS No.: 1550053-02-5

Molecular Formula: $C_{15}H_{14}FN_3O_2$

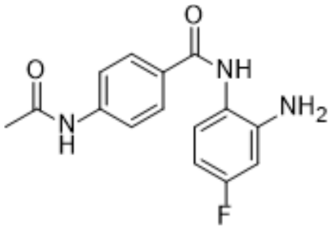
Molecular Weight: 287.29

Target: HDAC; HIV; Apoptosis

Pathway: Cell Cycle/DNA Damage; Epigenetics; Anti-infection; Apoptosis

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 : 250 mg/mL (870.20 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.4808 mL	17.4040 mL	34.8080 mL
	5 mM	0.6962 mL	3.4808 mL	6.9616 mL
	10 mM	0.3481 mL	1.7404 mL	3.4808 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: ≥ 2.08 mg/mL (7.24 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (7.24 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BRD3308 is a highly selective HDAC3 inhibitor with an IC_{50} of 54 nM. BRD3308 is 23-fold selectivity for HDAC3 over HDAC1 (IC_{50} of 1.26 μM) or HDAC2 (IC_{50} of 1.34 μM). BRD3308 suppresses pancreatic β-cell apoptosis induced by inflammatory cytokines or glucolipotoxic stress, and increases functional insulin release. BRD3308 activates HIV-1 transcription and disrupts HIV-1 latency[1][2][3].

IC_{50} & Target

HDAC3 54 nM (IC_{50})	HDAC3 29 nM (Ki)	HDAC1 1260 nM (IC_{50})	HDAC1 5100 nM (Ki)
HDAC2 1340 nM (IC_{50})	HDAC2 6300 nM (Ki)	HIV-1	

In Vitro

BRD3308 (5-30 μM; 6-24 hours) treatment increases HIV-1 expression in the 2D10 cell line[1].
?BRD3308 (15 μM; overnight) is able to induce outgrowth of HIV-1 from latently infected cells ex vivo in resting CD4+ T cells[1]
.?BRD3308 inhibits HDAC1, HDAC2 and HDAC3 with K_i values of 5.1 μM, 6.3 μM and 29 nM, respectively[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

RT-PCR[1]

Cell Line:	2D10 cells
Concentration:	5 μM, 10 μM, 15 μM, or 30 μM
Incubation Time:	6 hours, 12 hours, 18 hours, or 24 hours
Result:	An increase in HIV-1 expression was observed.

In Vivo

BRD3308 (5 mg/kg; intraperitoneal injection; every second day; male Zucker Diabetic Fatty rats) treatment reduces hyperglycaemia and increases insulin secretion in a rat model of type 2 diabetes. At the end of the hyperglycaemic clamp, circulating insulin levels are significantly higher in BRD3308-treated rats. Pancreatic insulin staining and contents are also significantly higher. BRD3308 preserves the functional β-cell mass against glucolipotoxicity in vivo[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Zucker Diabetic Fatty (Obese) rats (6-week-old)[2]
Dosage:	5 mg/kg
Administration:	Intraperitoneal injection; every second day
Result:	Reduced hyperglycaemia and increased insulin secretion in a rat model of type 2 diabetes.

REFERENCES

[1]. Barton KM, et al. Selective HDAC inhibition for the disruption of latent HIV-1 infection. PLoS One. 2014 Aug 19;9(8):e102684.

[2]. Lundh M, et al. Histone deacetylase 3 inhibition improves glycaemia and insulin secretion in obese diabetic rats. Diabetes Obes Metab. 2015 Jul;17(7):703-7.

[3]. Wagner FF, et al. An Isochemogenic Set of Inhibitors To Define the Therapeutic Potential of Histone Deacetylases in β-Cell Protection. ACS Chem Biol. 2016 Feb 19;11(2):363-74.

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