BRD-6929

Cat. No.: HY-100719

CAS No.: 849234-64-6

Molecular Formula: $C_{19}H_{17}N_3O_2S$

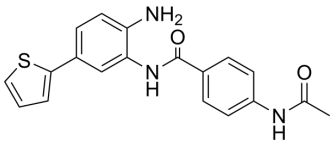
Molecular Weight: 351.42

Target: HDAC; HIV

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

Storage:

Powder	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 5 mg/mL (14.23 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8456 mL	14.2280 mL	28.4560 mL
	5 mM	0.5691 mL	2.8456 mL	5.6912 mL
	10 mM	0.2846 mL	1.4228 mL	2.8456 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.92 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BRD-6929 is a?potent, selective brain-penetrant inhibitor of class I histone deacetylase HDAC1 and HDAC2 inhibitor with IC_{50} of 1 nM and 8 nM, respectively. BRD-6929 shows high-affinity to HDAC1 and HDAC2 with?K_i?of 0.2 and 1.5 nM, respectively. BRD-6929 can be used for mood-related behavioral model research[3].

IC_{50} & Target

HDAC1	HDAC2	HDAC3	HIV-1
1 nM (IC_{50})	8 nM (IC_{50})	458 nM (IC_{50})	

In Vitro

In vitro?IC_{50} for HDAC1-9 by BRD-6929 using recombinant human HDAC enzymes and HDAC class-specific substrates. BRD-6929 and substrate are incubated for 180 min (HDAC1-3)?to control for HDAC1-3 inhibition, BRD-6929 is against HDAC1, HDAC2, HDAC3 and HDAC4-9 with IC_{50}s of 0.001 μM, 0.008 μM, 0.458 μM and >30 μM, respectively[1].

In vitro?binding affinity (K_i) and kinetics (half-life '$T_{1/2}$'?in minutes) for HDAC 1, 2 and 3 incubated with BRD-6929 (10 μM), the K_i values are <0.2 nM, 1.5nM, and 270 nM for HDAC 1, 2 and 3, respectively. The $T_{1/2}$ values are >2400 mins, >4800 mins, and 1200 mins for HDAC 1, 2 and 3, respectively[1].

	BRD-6929 (1 and 10 uM) does not cause an increase or decrease in overall cell number in brain region specific primary cultures. Additionally, BRD-6929 (10 uM) causes an increase in H4K12 acetylation in brain region specific primary cultures (striatum)[1]. BRD-6929 (1-10 uM; 6 hours) causes a significant increase in H2B acetylation in primary neuronal cell cultures. BRD-6929 (1-20 uM; 24 hours) induces a dose-dependent acetylation of H4K12ac with an EC_{50} of 7.2 μM in cultured neurons[1]. BRD-6929 potentiates the efficacy of gnidimacrin (a PKC Agonist) against latent HIV-1[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	BRD-6929 (intraperitoneal?injection; 45 mg/kg; single dose) exhibits a C_{max}, $T_{1/2}$ and AUC values of 17.7 μM, 7.2 hours, and 25.6 μM/L*hr, respectively in plasma. It shows a C_{max}, $T_{1/2}$ and AUC values of 0.83 μM, 6.4 hours, and 3.9 μM/L*hr, respectively in brain[1]. BRD-6929 (intraperitoneal?injection; 45 mg/kg; 10 days) acts as a deacetylase inhibitor in mouse brain. It significantly increases acetylation in each brain region by 1.5- to 2.0-fold compared to vehicle. The western blotting reveals that BRD-6929 significantly increases acetylation of histone H2B (tetra-acetylated), H3K9 and H4K12 in cortex, ventral striatum and hippocampus after the 10th daily treatment in adult male C57BL/6J mice[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Li-Huei Tsai, et al. Inhibition of hdac2 to promote memory. patent/US20120101147

[2]. Schroeder FA, et al. A selective HDAC 1/2 inhibitor modulates chromatin and gene expression in brain and altersmouse behavior in two mood-related tests. PLoS One. 2013 Aug 14;8(8):e71323.

[3]. Huang L, et al. Elimination of HIV-1 Latently Infected Cells by Gnidimacrin and a Selective HDAC Inhibitor. ACS Med Chem Lett. 2018 Feb 6;9(3):268-273.

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