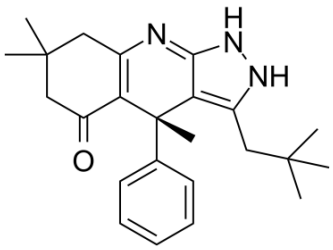
BRD3731

Cat. No.:	HY-124607B
CAS No.:	2056262-07-6
Molecular Formula:	$C_{24}H_{31}N_3O$
Molecular Weight:	377.52
Target:	GSK-3
Pathway:	PI3K/Akt/mTOR; Stem Cell/Wnt
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 : 50 mg/mL (132.44 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6489 mL	13.2443 mL	26.4887 mL
5 mM	0.5298 mL	2.6489 mL	5.2977 mL
10 mM	0.2649 mL	1.3244 mL	2.6489 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: ≥ 5 mg/mL (13.24 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 5 mg/mL (13.24 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

BRD3731 is a selective GSK3β inhibitor, with IC_{50}s of 15 nM and 215 nM for GSK3β and GSK3α, respectively. BRD3731 can be used for the research of a mood disorder, post-traumatic stress disorder (PTSD), psychiatric disorder, diabetes, and neurodegenerative disorder[1].

IC_{50} & Target

GSK-3β	GSK-3α
15 nM (IC_{50})	215 nM (IC_{50})

In Vitro

BRD3731 is a GSK3β- selective inhibitor extracted from patent US20160375006A1, compound example 272[1].
BRD3731 (1-10 μM; 24 hours) inhibits the phosphorylation of CRMP2 in SH-SY5Y cells[1].

	BRD3731 (20 μM; 24 hours) decreases β-catenin S33/37/T41 phosphorylation and induces β-catenin S675 phosphorylation inHL-60 cells[2]. BRD3731 (10-20 μM; 7-10 days) impairs colony formation in TF-1 and increases colony forming ability in the MV4–11 cell line [2] MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	BRD3731 (30 mg/kg; i.p.) reduces audiogenic seizures in Fmr1 KO mice[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Edward Scolnick, et al. Uses of paralog-selective inhibitors of gsk3 kinases. US20160375006A1.

[2]. Wagner FF, et, al. Exploiting an Asp-Glu "switch" in glycogen synthase kinase 3 to design paralog-selective inhibitors for use in acute myeloid leukemia. Sci Transl Med. 2018 Mar 7;10(431):eaam8460.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA