Alogliptin Benzoate

Cat. No.: HY-A0023

CAS No.: 850649-62-6

Molecular Formula: $C_{25}H_{27}N_5O_4$

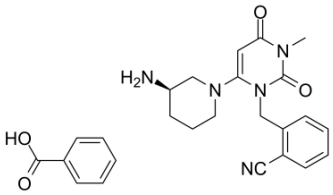
Molecular Weight: 461.51

Target: Dipeptidyl Peptidase; Ferroptosis

Pathway: Metabolic Enzyme/Protease; 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 : 50 mg/mL (108.34 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.1668 mL	10.8340 mL	21.6680 mL
5 mM	0.4334 mL	2.1668 mL	4.3336 mL
10 mM	0.2167 mL	1.0834 mL	2.1668 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: ≥ 2.5 mg/mL (5.42 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.42 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.42 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Alogliptin Benzoate (SYR-322) is a potent, selective and orally active inhibitor of DPP-4 with an IC_{50} of <10 nM, and exhibits greater than 10,000-fold selectivity over DPP-8 and DPP-9. Alogliptin Benzoate can be used for the research of type 2 diabetes[1][2][3].

IC_{50} & Target

IC50: <10 nM (DPP-4)[1]

In Vitro

Alogliptin (1 nM; 5-60 min) inhibits LPS-induced extracellular signal-regulated kinase (ERK) phosphorylation in U937 cells[2].

	Alogliptin (0.5-5 nM; 24 h) inhibits LPS-stimulated MMP-1 secretion and mRNA expression that is mediated by ERK pathway in U937 cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Alogliptin (0.01-1 mg/kg; p.o.) produced dose-dependent improvements in glucose tolerance and increased plasma insulin levels in female Wistar fatty rats[1]. Alogliptin (40 mg/kg/day for 2 weeks; p.o.) reduces infarction area and improves brain vascular integrity in middle cerebral artery occlusion (MCAO) mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Sci Rep. 2019 Dec 2;9(1):18094.
- Biochem Biophys Res Commun. 2019 Apr 2;511(2):387-393.
- Chromatography. 2015,36(1):19-24.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Feng J, et, al. Discovery of alogliptin: a potent, selective, bioavailable, and efficacious inhibitor of dipeptidyl peptidase IV. J Med Chem. 2007 May 17;50(10):2297-300.

[2]. Ta NN, et, al. DPP-4 (CD26) inhibitor alogliptin inhibits TLR4-mediated ERK activation and ERK-dependent MMP-1 expression by U937 histiocytes. Atherosclerosis. 2010 Dec;213(2):429-35.

[3]. Hao FL, et, al. The neurovascular protective effect of alogliptin in murine MCAO model and brain endothelial cells. Biomed Pharmacother. 2019 Jan;109:181-187.

[4]. Asakawa T, et, al. A novel dipeptidyl peptidase-4 inhibitor, alogliptin (SYR-322), is effective in diabetic rats with sulfonylurea-induced secondary failure. Life Sci. 2009 Jul 17;85(3-4):122-6.

[5]. Moritoh Y, et al. The dipeptidyl peptidase-4 inhibitor alogliptin in combination with pioglitazone improves glycemic control, lipid profiles, and increases pancreatic insulin content in ob/ob mice. Eur J Pharmacol. 2009 Jan 14;602(2-3):448-54.

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