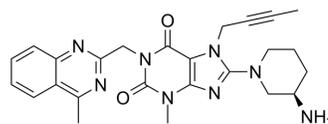


Linagliptin

Cat. No.:	HY-10284		
CAS No.:	668270-12-0		
Molecular Formula:	C ₂₅ H ₂₈ N ₈ O ₂		
Molecular Weight:	472.54		
Target:	Dipeptidyl Peptidase; Autophagy; Ferroptosis		
Pathway:	Metabolic Enzyme/Protease; Autophagy; Apoptosis		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro	DMSO : 25 mg/mL (52.91 mM); ultrasonic and warming and heat to 80°C				
		Solvent Concentration	Mass 1 mg	5 mg	10 mg
	Preparing Stock Solutions	1 mM	2.1162 mL	10.5811 mL	21.1622 mL
		5 mM	0.4232 mL	2.1162 mL	4.2324 mL
10 mM		0.2116 mL	1.0581 mL	2.1162 mL	
Please refer to the solubility information to select the appropriate solvent.					
In Vivo	<ol style="list-style-type: none"> Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 25 mg/mL (52.91 mM); Clear solution Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: 2.5 mg/mL (5.29 mM); Clear solution; Need ultrasonic Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.5 mg/mL (5.29 mM); Clear solution 				

BIOLOGICAL ACTIVITY

Description	Linagliptin is a highly potent, selective DPP-4 inhibitor with IC ₅₀ of 1 nM.
IC₅₀ & Target	IC ₅₀ : 1 nM (DPP-4)
In Vitro	Linagliptin inhibits DPP-4 activity in vitro in several independent experiments with IC ₅₀ values of 0.4, 0.5, 0.9, and 1.1 nM (mean IC ₅₀ , approximately 1 nM). Linagliptin inhibits FAP with an IC ₅₀ of 89 nM (approximately 90-fold selectivity versus DPP-4) ^[2] .

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

In Vivo

In male Wistar rats, Beagle dogs, and Rhesus monkeys, xanthine linagliptin proves to be a highly efficacious, long-lasting, and potent DPP-4 inhibitor providing >70% inhibition for >7 h for all three species after oral administration of 1 mg/kg. Single oral administration of linagliptin to db/db mice 45 min prior to an oral glucose tolerance test reduced plasma glucose excursion in a dose-dependent manner from 0.1 mg/kg (15% inhibition) to 1 mg/kg (66% inhibition)^[1]. Linagliptin (3 and 10 mg/kg) dose-dependently inhibits the DPP-4 enzyme in plasma within 30 min of administration. Linagliptin (1 mg/kg, p.o.) significantly reduces glucose excursion by approximately 50%^[2]. Oral administration of the DPP-4 inhibitor linagliptin (3 mg/kg, p.o.) strongly reduces DPP-4 activity, stabilizes active GLP-1 in chronic wounds, and improves healing in ob/ob mice. At day 10 postwounding, linagliptin-treated ob/ob mice show largely epithelialized wounds characterized by the absence of neutrophils^[3].

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

PROTOCOL

Kinase Assay ^[3]

EDTA plasma (20 μ L) is diluted with 30 μ L of DPP-4 assay buffer (100 mM Tris and 100 mM NaCl, adjusted to pH 7.8 with HCl) and mixed with 50 μ L of H-Ala-Pro-7-amido-4-trifluoromethylcoumarin. The 200 mM stock solution in dimethylformamide is diluted 1:1000 with water to yield a final concentration of 100 μ M. The plate is incubated at room temperature for 10 min, and fluorescence in the wells is determined by using a Victor 1420 Multilabel Counter at an excitation wavelength of 405 nm and an emission wavelength of 535 nm. For the detection of DPP-4 activity in wound lysates, 100 μ g of protein from the respective wound lysates are used instead of 20 μ L of plasma. Active GLP-1 is also detected from 100 μ g of respective wound tissue samples and analyzed by using the Mouse/Rat Total Active GLP-1 Assay Kit.

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

Cell Assay ^[3]

A total of 4.0×10^7 keratinocytes per well are seeded into 24-well plates. After reaching 50% confluence, cells are starved for 24 h with DMEM. Proliferation of cells is assessed by using 1 μ Ci/mL of [³H]methyl-thymidine in DMEM in the presence of 10% fetal bovine serum and increasing concentrations of linagliptin (3, 30, 300, or 600 nM) for 24 h. Cells are then washed twice with phosphate-buffered saline and incubated in 5% trichloroacetic acid at 4°C for 30 min, and the DNA is solubilized in 0.5 mol/L NaOH for 30 min at 37°C. Finally, [³H]thymidine incorporation is determined.

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

Animal Administration ^[3]

Each experimental group (vehicle or linagliptin treatment) consists of 10 individual ob/ob mice (n=10). Animals are treated orally once a day (8:00 AM) by gastrogavage using vehicle (1% methylcellulose) or linagliptin (3 mg/kg body weight in 1% methylcellulose) beginning 2 days (day-2) before wounding. After wounding, animals are subsequently treated once a day throughout the 10-day healing period.

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

CUSTOMER VALIDATION

- Cell Rep. 2023 Feb 28.
- Neural Regen Res. 2022.
- Biochem Pharmacol. 2018 Oct;156:312-321.
- Int J Mol Sci. 2024 Mar 8, 25(5), 3008.
- Molecules. 2022 Apr 12;27(8):2478.

See more customer validations on www.MedChemExpress.com

REFERENCES

- [1]. Eckhardt M, et al. 8-(3-(R)-aminopiperidin-1-yl)-7-but-2-ynyl-3-methyl-1-(4-methyl-quinazolin-2-ylmethyl)-3,7-dihydropurine-2,6-dione (BI 1356), a highly potent, selective, long-acting, and orally bioavailable DPP-4 inhibitor for the treatment of type 2 d
- [2]. Thomas L, et al. (R)-8-(3-amino-piperidin-1-yl)-7-but-2-ynyl-3-methyl-1-(4-methyl-quinazolin-2-ylmethyl)-3,7-dihydro-purine-2,6-dione (BI 1356), a novel xanthine-based dipeptidyl peptidase 4 inhibitor, has a superior potency and longer duration of action
- [3]. Schurmann C, et al. The dipeptidyl peptidase-4 inhibitor linagliptin attenuates inflammation and accelerates epithelialization in wounds of diabetic ob/ob mice. *J Pharmacol Exp Ther.* 2012 Jul;342(1):71-80.
- [4]. Huan Y, et al. The dual DPP4 inhibitor and GPR119 agonist HBK001 regulates glycemic control and beta cell function ex and in vivo. *Sci Rep.* 2017 Jun 28;7(1):4351.
-

Caution: Product has not been fully validated for medical applications. For research use only.

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