Vincamine

Cat. No.: HY-B1021

CAS No.: 1617-90-9

Molecular Formula: $C_{21}H_{26}N_2O_3$

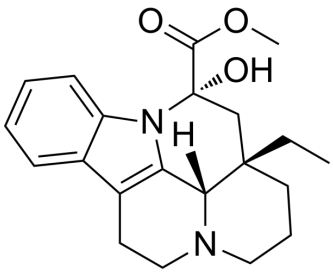
Molecular Weight: 354.44

Target: Free Fatty Acid Receptor

Pathway: GPCR/G Protein

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 (70.53 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.8214 mL	14.1068 mL	28.2135 mL
5 mM	0.5643 mL	2.8214 mL	5.6427 mL
10 mM	0.2821 mL	1.4107 mL	2.8214 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 (7.05 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.05 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Vincamine is a monoterpenoid indole alkaloid extracted from the Madagascar periwinkle. Vincamine is a peripheral vasodilator and exerts a selective vasoregulator action on the brain microcapilar circulation[1]. Vincamine is a GPR40 agonist and acts as a β-cell protector by ameliorating β-cell dysfunction and promoting glucose-stimulated insulin secretion (GSIS). Vincamine improves glucose homeostasis in vivo, and has the potential for the type 2 diabetes mellitus (T2DM) research[2].

In Vitro

Vincamine (20, 40 and 80 μM; 24 hours) exerts a significant, concentration-dependent protective effect in LPS-treated human corneal epithelial cells (HCECs) cells[1].
Vincamine (20, 40 and 80 μM; 24 hours) significantly reduces ROS level in a dose-dependent manner in LPS-treated human corneal epithelial cells (HCECs)cells. Additionally, after Vincamine administration, the levels of MDA is also significantly

reduced while the levels of T-AOC, and SOD are increased in a dose-dependent manner[1].
Vincamine (20, 40 and 80 μM; 24 hours) rescues TrxR activity in a dose-dependent manner in HCECs. However, the intracellular activities of Trx, GR and GPx are neither inhibited nor activated by both LPS and Vincaminer[1].
Vincamine could activate GPR40 (EC_{50}=6.28 μM) with DHA (GPR40 ligand) as a positive control (EC_{50}=3.85 μM) in hGPR40-CHO cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Vincamine (intraperitoneal injection; 15 and 30 mg/kg/day; 6 weeks) improves glucose tolerance in type 2 diabetic model mice. It effectively lowers the levels of fasting blood glucose and glycated hemoglobin. At the same time, it ameliorates oral glucose tolerance and elevated glucose-induced plasma insulin concentration without influence on basal insulin secretion in vivo[2].<.br>
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male and female db/db mice (BKS.Cg-Dock$^{7m+/+}$Lepr^{db}/J) and HFD/STZ-induced type 2 diabetic model mice
Dosage:	15 and 30 mg/kg/day
Administration:	Intraperitoneal injection; 15 and 30 mg/kg/day; 6 weeks
Result:	Enhanced glucose tolerance in HFD/STZ and db/db male mice.

REFERENCES

[1]. Te Du, et al. Vincamine as a GPR40 agonist improves glucose homeostasis in type 2 diabetic mice. J Endocrinol. 2019 Feb 1;240(2):195-214.

[2]. Li Wu, et al. Vincamine prevents lipopolysaccharide induced inflammation and oxidative stress via thioredoxin reductase activation in human corneal epithelial cells. Am J Transl Res. 2018 Jul 15;10(7):2195-2204. eCollection

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