KY-226

Cat. No.: HY-120327

CAS No.: 1621673-53-7

Molecular Formula: $C_{27}H_{31}NO_3S_2$

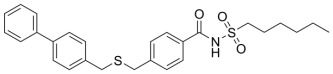
Molecular Weight: 482

Target: Phosphatase

Pathway: Metabolic Enzyme/Protease

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 : 250 mg/mL (518.67 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.0747 mL	10.3734 mL	20.7469 mL
5 mM	0.4149 mL	2.0747 mL	4.1494 mL
10 mM	0.2075 mL	1.0373 mL	2.0747 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: ≥ 2.08 mg/mL (4.32 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.32 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.32 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

KY-226 is a potent, selective, orally active and allosteric protein tyrosine phosphatase 1B (PTP1B) inhibitor with an IC_{50} of 0.25 μM, and without PPARγ agonist activity. KY-226 exerts anti-diabetic and anti-obesity effects by enhancing insulin and leptin signaling, respectively. KY-226 also protects neurons from cerebral ischemic injury[1][2].

IC_{50} & Target

IC50: 0.25 μM (Protein tyrosine phosphatase 1B (PTP1B))[2]

In Vitro

In human hepatoma-derived cells (HepG2), KY-226 (0.3-10 μM) increases the phosphorylated insulin receptor (pIR) produced

by insulin[1].
KY-226 (1 μM; 24 hours; bEnd.3 cells) treatment rescues lipopolysaccharide-induced reduction of mRNA and protein levels of ZO-1. KY-226 treatment restores phosphorylation of pAkt (T308) and its downstream target forkhead box protein O1 (FoxO1) (S256) in bEnd.3 cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	bEnd.3 cells stimulated with LPS
Concentration:	1 μM
Incubation Time:	24 hours
Result:	Rescued lipopolysaccharide-induced reduction of mRNA and protein levels of ZO-1.

In Vivo

KY-226 (10-30 mg/kg/day; oral administration; daily; for 4 weeks; male db/db mice) treatment significantly reduces plasma glucose and triglyceride levels as well as hemoglobin A1c values without increasing body weight gain[1].
KY-226 attenuates plasma glucose elevations in the oral glucose tolerance test. KY-226 also increases pIR and phosphorylated Akt in the liver and femoral muscle[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male db/db mice (8-11 weeks old)[1]
Dosage:	10 mg/kg and 30 mg/kg
Administration:	Oral administration; daily; for 4 weeks
Result:	Significantly reduced plasma glucose and triglyceride levels as well as hemoglobin A1c values without increasing body weight gain.

REFERENCES

[1]. Ito Y, et al. Therapeutic effects of the allosteric protein tyrosine phosphatase 1B inhibitor KY-226 on experimental diabetes and obesity via enhancements in insulin and leptin signaling in mice. J Pharmacol Sci. 2018 May;137(1):38-46.

[2]. Sun M, et al. KY-226 Protects Blood-brain Barrier Function Through the Akt/FoxO1 Signaling Pathway in Brain Ischemia. Neuroscience. 2019 Feb 10;399:89-102.

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