Epalrestat

Cat. No.: HY-66009

CAS No.: 82159-09-9

Molecular Formula: $C_{15}H_{13}NO_3S_2$

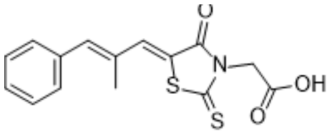
Molecular Weight: 319.4

Target: Aldose Reductase

Pathway: Metabolic Enzyme/Protease

Storage: -20°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 20 mg/mL (62.62 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.1309 mL	15.6544 mL	31.3087 mL
	5 mM	0.6262 mL	3.1309 mL	6.2617 mL
	10 mM	0.3131 mL	1.5654 mL	3.1309 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2 mg/mL (6.26 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Epalrestat is an orally active aldose reductase inhibitor that acts on diabetic neuropathy[1][2][3].

In Vitro

Epalrestat (100 and 200 μM, 24 h) inhibits cell viability and induces cell apoptosis in rat Schwann cells (SCs)[5].

Epalrestat (10 and 50 μM, 24 h) increases intracellular glutathione levels in rat SCs by up-regulating γ-GCS via Nrf2 activation [5].

Epalrestat (50 μM, 16 h) protects SCs from oxidative stress[5].

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

RT-PCR[5]

Cell Line:	rat SCs
Concentration:	10 or 50 μM
Incubation Time:	4 h

Result:	Increased the nuclear level of active Nrf2 by 1.8- and 3.8-fold at 10 and 50 μM, and failed to increase Nrf2 mRNA levels.

In Vivo

Epalrestat (0.08% (w/w) in regular chow, 8 weeks) shows nephritic protective effect in diabetic nephropathy in db/db mice[4].

Epalrestat (100 mg/kg, i.g., daily for 6 weeks) protects rats from diabetic peripheral nerve injury in diabetic peripheral neuropathy (DPN) model induced by Streptozotocin (HY-13753)[6].

Epalrestat (50 mg/kg, oral gavage, twice a day) reduces cerebral ischemia-induced infarct volume and BBB permeability in mice[7].

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

Animal Model:	db/db mice[4]
Dosage:	0.08% (w/w) in fed regular chow
Administration:	8 weeks
Result:	Ameliorated GBM thickening and mesangial matrix deposition in kidney tissue. Reduced the elevated sorbitol and fructose in the plasma, urine, and renal cortex of db/db mice. Reduced myo-inositol in the plasma and urine, whereas increased myo-inositol in the renal cortex.

Animal Model:	Rats were treated with high-fat and high-sugar diet for 4 weeks, and injected with Streptozotocin at 4 and 8 weeks[6]
Dosage:	100 mg/kg/d
Administration:	i.g. 6 weeks
Result:	Improved pathological structures of neurites and myelin. Increased SOD, CAT and GPX protein levels in sciatic nerves. Decrease aldose reductase level in sciatic nerves.

CUSTOMER VALIDATION

- J Transl Med. 2023 Oct 7;21(1):700.

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REFERENCES

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[3]. Li QR, et al. Epalrestat protects against diabetic peripheral neuropathy by alleviating oxidative stress and inhibiting polyol pathway. Neural Regen Res. 2016 Feb;11(2):345-51.

[4]. Zhang T, et al. The Aldose Reductase Inhibitor Epalrestat Maintains Blood-Brain Barrier Integrity by Enhancing Endothelial Cell Function during Cerebral Ischemia. Mol Neurobiol. 2023 Jul;60(7):3741-3757.

[5]. Ramirez, M.A. and N.L. Borja, Epalrestat: an aldose reductase inhibitor for the treatment of diabetic neuropathy. Pharmacotherapy, 2008. 28(5): p. 646-55.

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[7]. Hotta, N., et al., Clinical investigation of epalrestat, an aldose reductase inhibitor, on diabetic neuropathy in Japan: multicenter study. Diabetic Neuropathy Study Group in Japan. J Diabetes Complications, 1996. 10(3): p. 168-72.

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

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