Hirudin

Cat. No.: HY-P2813

CAS No.: 8001-27-2

Target: Thrombin; Apoptosis

Pathway: Metabolic Enzyme/Protease; Apoptosis

Storage: -80°C

Hirudin

BIOLOGICAL ACTIVITY

Description

Hirudin is a thrombin inhibitor with blood anticoagulant property. Hirudin has potent anti-thrombotic, wound repair, anti-fibrosis, anti-tumor and anti-hyperuricemia effects. Hirudin also affects diabetic complications, cerebral hemorrhage, and others[1].

In Vitro

Hirudin inhibits the activity of thrombin, deprives the ability of thrombin to cleave fibrinogen, prevents the formation of fibrin and the cross-linking polymerization process of fibrin monomer in internal and external coagulation pathway[1].
Hirudin reduces cell apoptosis of human microvascular endothelial cells (HMVECs) and suppresses the expression of p-JAK2 via antagonizing thrombin[1].
Hirudin inhibits VEGF-Notch pathway and cell proliferation of HMVECs at high doses[1].
Hirudin (3-10 mg/mL) reverses the abnormal proliferation and fibrosis in HK-2 cells caused by TGF-β1[1].
Hirudin depresses the myocardial fibroblasts induced by angiotensin II by dose-dependently inhibits oxidative stress, regulates fibrosis-related factors, and represses the ERK1/2 pathway[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Hirudin increases the viability of rat random skin flap and reduces inflammatory responses[1].
Hirudin promotes the wound healing in SD rats after laser surgery[1].
Hirudin (10 and 15 mg/kg; i.g. once daily for 21 days) improves renal interstitial fibrosis to reduce renal tubule injury and inflammation in unilateral ureteral obstruction (UUO) mice[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male balb/c mice with underwent unilateral ureteral ligation (UUO)[2]
Dosage:	10 and 15 mg/kg
Administration:	Oral gavage; 10 and 15 mg/kg, once daily for 21 days
Result:	Reduced renal damages and suppressed the upregulation of α-SMA, collagen deposition in UUO mice. Increased the level of fibrosis (collagen-I, FN, α-SMA), N-cad, slug and E-cad in UUO mice. Decreased the level of IL-1β, IL-6 and TNF-α, apoptosis of renal tubular cells in UUO mice. Decreased the expression of inflammatory factors, the occurrence of EMT, the incidence of fibrosis and the apoptosis of TGF-β-induced renal tubular epithelial cell.

REFERENCES

[1]. Junren C,et al. Pharmacological Activities and Mechanisms of Hirudin and Its Derivatives - A Review. Front Pharmacol. 2021 Apr 16;12:660757.

[2]. Xie Y, et al. Hirudin improves renal interstitial fibrosis by reducing renal tubule injury and inflammation in unilateral ureteral obstruction (UUO) mice. Int Immunopharmacol. 2020 Apr;81:106249.

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