Xanthine oxidase-IN-6

Cat. No.: HY-146560

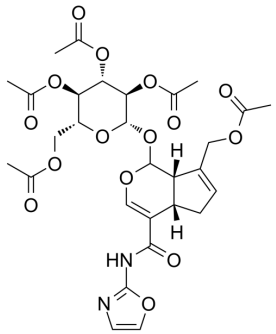
Molecular Formula: $C_{29}H_{34}N_2O_{15}$

Molecular Weight: 650.58

Target: Xanthine Oxidase; NF-κB; Toll-like Receptor (TLR); TNF Receptor

Pathway: Metabolic Enzyme/Protease; NF-κB; Immunology/Inflammation; Apoptosis

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Xanthine oxidase-IN-6 (Compound 6c) is a potent, orally active, mixed-type xanthine oxidase (XOD) inhibitor with an IC_{50} value of 1.37 μM. Xanthine oxidase-IN-6 shows strong anti-hyperuricemia and renal protective activity[1].

IC_{50} & Target			
XOD 1.37 μM (IC_{50})	NF-κB	TLR4	TNF-α

In Vitro

Xanthine oxidase-IN-6 (Compound 6c) is a mixed-type XOD inhibitor, preferentially bound to the free enzyme and not the enzyme substrate complex[1].

Xanthine oxidase-IN-6 is stable in simulated gastrointestinal digestion, with hydrolysis half-life more than 4 h[1].

Xanthine oxidase-IN-6 (0-100 μM) exhibits an obvious anti-inflammatory effect by reducing the level of inflammatory factors (TGF-β, TNF-α and IL-1β) in a dose-dependent manner[1].

Xanthine oxidase-IN-6 (0-100 μM, 48 h) inhibits HK-2 cell epithelial mesenchymal transition under high level of uric acid[1].

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

Western Blot Analysis[1]

Cell Line:	HK-2 cells
Concentration:	12.5, 25, 50, and 100 μM
Incubation Time:	48 h
Result:	Reduced the protein levels of α-SMA and Collagen I in a dose-dependent manner

In Vivo

Xanthine oxidase-IN-6 (Compound 6c) (0-20 mg/kg; i.g.; once daily for 2 weeks) shows anti-hyperuricemic effects, alleviates kidney damage, and inhibits XOD activity in a dose-dependent manner[1].

Xanthine oxidase-IN-6 (0-20 mg/kg; i.g.; once daily for 2 weeks) effectively reduces renal fibrosis and inflammation[1].

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

Animal Model:	Kunming mice (male, weight 20 ± 2 g). Hyperuricemic mouse model established by administering 0.5% CMC-Na (10 mL/kg), adenine (200 mg/kg) and potassium oxonate (500 mg/kg); oral gavage once daily for 2 or 4 weeks[1].
Dosage:	5, 10 and 20 mg/kg

Administration:	Gavage, once daily for 2 weeks
Result:	Effectively decreased the levels of SUA, Cr, and BUN, effectively inhibited XOD activity and urate accumulation in a dose-dependent manner. Remarkedly improved the morphologic lesions with less fibrosis in the interstitium. Reduced the production of multiple cytokines (TNF-α, IL-8, and IL-1β). Reduced the expression of α-SMA, collagen I, TLR4, NF-κB, IκBα and TNF-α.

REFERENCES

[1]. Jia-shu Chen, et al. Synthesis and biological evaluation of geniposide derivatives as inhibitors of hyperuricemia, inflammatory and fibrosis. Eur J Med Chem. 2022 Apr 20;237:114379.

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