

Product Data Sheet

Product Name: Rehmannioside A

Cat. No.: GC33468

Chemical Properties

Cas. No. 81720-05-0

SMILES OCC1(C(C2OC(C(C(O)C3O)O)OC3COC(C(C(O)C4O)O)OC4CO)C(C=CO2)C5O)C5O1

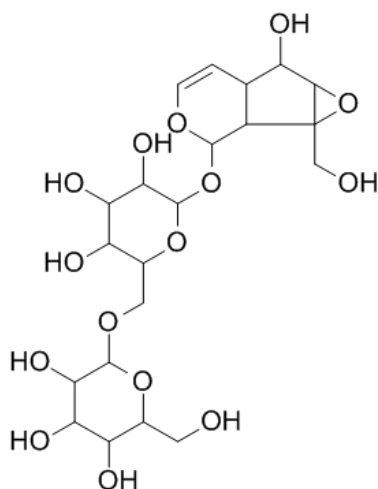
Formula $C_{21}H_{32}O_{15}$ M.Wt 524.47

Solubility DMSO: 200 mg/mL (680.7 mM),
Need ultrasonic Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines MC3T3-E1 cells

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

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Preparation Method MC3T3-E1 cells were seeded into 96-well plates and treated with Rehmannioside A (10, 25, 50, 100, 150, 200, and 300 μ M) for 24h. For Dexamethasone treatment, the cells were pretreated with Rehmannioside A for 12h. After 24h of treatment, the cells were washed twice with phosphate-buffered saline and incubated with CCK-8 working solution for 2h at 37°C. The optical density was measured at 450nm using a microplate reader.

Reaction Conditions 10, 25, 50, 100, 150, 200, 300 μ M; 24h

Applications Dexamethasone significantly decreased cell viability at 24h, while Rehmannioside A at 50-200 μ M significantly improved the cell viability of osteoblast exposed to Dexamethasone.

Animal experiment [2]:

Animal models Sprague-Dawley (SD) rats

Preparation Method Rats were anesthetized using chloral hydrate (350mg/kg, i.p.). Animals were randomly divided into 4 groups (n=20/group): sham-operated rats received the same surgical procedure without the occlusion of double ligation; both carotid arteries were gently separated from the vagus nerve, and permanent artery occlusion was implemented with silk thread. For verification, rats in Vascular dementia (VD) group were randomly subdivided into a control VD Group treated with PBS (VD group), and a 40mg/kg Rehmannioside A(ReA)+VD (ReA-L group) and a 80mg/kg Rehmannioside A+VD (ReA-H group). The sham group consisted of sham-operated rats were treated with PBS (Sham group). RehA was injected intraperitoneally to rats daily for 14 consecutive days.

Dosage form 40, 80mg/kg; 14 days; i.p.

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Applications

Vascular dementia (VD) rats exhibited higher percentage of TUNEL and active Caspase-3 positive cells. Rehmannioside A dose-dependently reduced TUNEL and active Caspase-3 expressions. Rehmannioside A could reduce the over-expression of cleaved Caspase-3, PARP, and Cyto-c in the hippocampus of VD rats.

References:

- [1] Huang H, Ji F, Qi G, et al. Rehmannioside A promotes the osteoblastic differentiation of MC3T3-E1 cells via the PI3K/AKT signaling pathway and inhibits glucocorticoid-induced bone loss in vivo[J]. Journal of Pharmacological Sciences, 2024, 156(4): 247-257.
- [2]Sun M, Shen X, Ma Y. Rehmannioside A attenuates cognitive deficits in rats with vascular dementia (VD) through suppressing oxidative stress, inflammation and apoptosis[J]. Biomedicine & Pharmacotherapy, 2019, 120: 109492.

Background

Rehmannioside A is an inhibitor of cytochrome P450 enzymes (CYP3A4, 2C9, and 2D6), with IC₅₀ values of 10.08, 12.62, and 16.43μM, respectively^[1]. Rehmannioside A is an iridoid glycoside isolated from *Rehmanniae radix*, possessing various biological activities including anti-inflammatory, antioxidant, anti-apoptotic, and neuroprotective effects^[2, 3].

In vitro, treatment of mouse osteoblast cell lines (MC3T3-E1 cells) with Rehmannioside A (10-300μM) for 24h significantly improved the survival rate of dexamethasone-exposed osteoblasts at 50-200μM, and promoted the activation of the PI3K/AKT signaling pathway in MC3T3-E1 cells^[4]. Treatment of HK2 cells with Rehmannioside A (20-80μM) for 24h significantly improved the survival rate of high glucose (HG)-induced HK2 cells, inhibited apoptosis and oxidative stress, and suppressed the activation of the intracellular MAPK pathway^[5].

In vivo, Rehmannioside A (40, 80mg/kg) administered intraperitoneally to rats with vascular dementia (VD) for 14 days dose-dependently reduced the overexpression of TUNEL-positive cells, active Caspase-3, PARP, and cytochrome c in the hippocampus^[6]. Oral administration of Rehmannioside A (5, 15, 45mg/kg) to 5xFAD mice significantly improved learning, memory, and cognitive function, reduced the accumulation of Aβ plaques in the hippocampus, reduced the number of microglia,

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increased the expression of PSD 95 and synaptophysin-1 proteins, and alleviated oxidative stress and inflammation^[7].

References:

- [1] Wang C, Zhou N, Li M, et al. Rehmannioside A inhibits the activity of CYP3A4, 2C9 and 2D6 in vitro[J]. *Xenobiotica*, 2024, 54(4): 195-200.
- [2] Zhao K, Zhu H, Zheng L, et al. Harnessing the multi-targeted potential of rehmanniae radix natural products against renal fibrosis: a mechanistic review[J]. *Frontiers in Pharmacology*, 2025, 16: 1690036.
- [3] Zhong H Z, Mo J, Li Y X, et al. Changes in Rehmanniae Radix processing and their impact on ovarian hypofunction: potential mechanisms of action[J]. *Frontiers in pharmacology*, 2024, 15: 1426972.
- [4] Huang H, Ji F, Qi G, et al. Rehmannioside A promotes the osteoblastic differentiation of MC3T3-E1 cells via the PI3K/AKT signaling pathway and inhibits glucocorticoid-induced bone loss in vivo[J]. *Journal of Pharmacological Sciences*, 2024, 156(4): 247-257.
- [5] Huai L, Yang L, Xu Q, et al. Rehmannioside A protects against high glucose-induced apoptosis and oxidative stress of renal tubular epithelial cells by inhibiting the MAPK pathway[J]. *Tropical Journal of Pharmaceutical Research*, 2021, 20(8): 1553-1558.
- [6] Sun M, Shen X, Ma Y. Rehmannioside A attenuates cognitive deficits in rats with vascular dementia (VD) through suppressing oxidative stress, inflammation and apoptosis[J]. *Biomedicine & Pharmacotherapy*, 2019, 120: 109492.
- [7] Lai Y, Zhao H. Therapeutic efficacy of rehmannioside A on 5× FAD mice in Alzheimer's disease[J]. *Scientific Reports*, 2025, 15(1): 39825.

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