Hydrochlorothiazide

Cat. No.: HY-B0252

CAS No.: 58-93-5

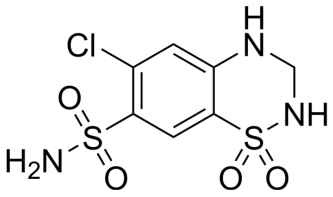
Molecular Formula: $C_7H_8ClN_3O_4S_2$

Molecular Weight: 297.74

Target: TGF-beta/Smad; Potassium Channel

Pathway: Stem Cell/Wnt; TGF-beta/Smad; Membrane Transporter/Ion Channel

Storage: Powder -20°C 3 years
4°C 2 years
* The compound is unstable in solutions, freshly prepared is recommended.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (167.93 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.3586 mL	16.7932 mL	33.5864 mL
	5 mM	0.6717 mL	3.3586 mL	6.7173 mL
	10 mM	0.3359 mL	1.6793 mL	3.3586 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: ≥ 2.5 mg/mL (8.40 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.40 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.40 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Hydrochlorothiazide (HCTZ), an orally active diuretic agent of the thiazide class, inhibits transforming TGF-β/Smad signaling pathway. Hydrochlorothiazide has direct vascular relaxant effects via opening of the calcium-activated potassium (KCA) channel. Hydrochlorothiazide improves cardiac function, reduces fibrosis and has antihypertensive effect[1][2][3].

In Vitro

Hydrochlorothiazide belongs to thiazide class of diuretics. It reduces blood volume by acting on the kidneys to reduce sodium (Na) reabsorption in the distal convoluted tubule. The major site of action in the nephron appears on an electroneutral Na^+-Cl co-transporter by competing for the chloride site on the transporter. By impairing Na transport in the distal convoluted tubule, hydrochlorothiazide induces a natriuresis and concomitant water loss. Thiazides increase the

	reabsorption of calcium in this segment in a manner unrelated to sodium transport. Additionally, by other mechanisms, Hydrochlorothiazide is believed to lower peripheral vascular resistance[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Hydrochlorothiazide (HCTZ; orally bygavage; 12.5 mg/kg/d; 8 weeks) has improved cardiac function, reduced cardiac interstitial fibrosis and collagen volume fraction, decreased expression of AT1, TGF-β and Smad2 in the cardiac tissues in adult male Sprague Dawley rats. In addition, hydrochlorothiazide reduces plasma angiotensin II and aldosterone levels. Furthermore, hydrochlorothiazide inhibits angiotensin II-induced TGF-β1 and Smad2 protein expression in the neonatal rat ventricular fibroblasts[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Hypertension. 2021 Jan 25;HYPERTENSIONAHA12015636.
- J Orthop Surg Res. 2024 Feb 19;19(1):147.

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REFERENCES

[1]. Duarte, J.D. and R.M. Cooper-DeHoff, Mechanisms for blood pressure lowering and metabolic effects of thiazide and thiazide-like diuretics. Expert Rev Cardiovasc Ther, 2010. 8(6): p. 793-802.

[2]. Magdy M Abdelquader, et al. Inhibition of Co-Crystallization of Olmesartan Medoxomil and Hydrochlorothiazide for Enhanced Dissolution Rate in Their Fixed Dose Combination. AAPS PharmSciTech. 2018 Dec 17;20(1):3.

[3]. Jinghong Luo, et al. Hydrochlorothiazide modulates ischemic heart failure-induced cardiac remodeling via inhibiting angiotensin II type 1 receptor pathway in rats. Cardiovasc Ther. 2017 Apr;35(2).

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