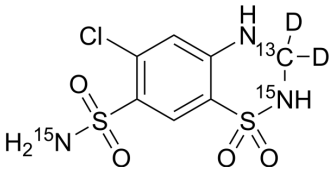
Hydrochlorothiazide-$^{15}N_2$,^{13}C,d_2

Cat. No.:	HY-B0252S3
CAS No.:	2140316-81-8
Molecular Formula:	$C_6{}^{13}CH_6D_2ClN^{15}N_2O_4S_2$
Molecular Weight:	302.73
Target:	Potassium Channel; TGF-beta/Smad; Isotope-Labeled Compounds
Pathway:	Membrane Transporter/Ion Channel; Stem Cell/Wnt; TGF-beta/Smad; Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Hydrochlorothiazide-$^{15}N_2$,^{13}C,d_2 is ^{15}N and deuterated labeled Hydrochlorothiazide (HY-B0252). 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	Stable heavy isotopes of hydrogen, carbon, and other elements have been incorporated into drug molecules, largely as tracers for quantitation during the drug development process. Deuteration has gained attention because of its potential to affect the pharmacokinetic and metabolic profiles of drugs[1]. 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[2]. 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[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. 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.

[2]. 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.

[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).

[4]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. Ann Pharmacother. 2019 Feb;53(2):211-216.

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