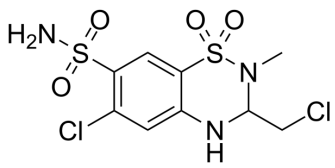
Methyclothiazide

Cat. No.:	HY-B0562
CAS No.:	135-07-9
Molecular Formula:	$C_9H_{11}Cl_2N_3O_4S_2$
Molecular Weight:	360.24
Target:	Carbonic Anhydrase
Pathway:	Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 72 mg/mL (199.87 mM; Need ultrasonic and warming)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.7759 mL	13.8796 mL	27.7593 mL
5 mM	0.5552 mL	2.7759 mL	5.5519 mL
10 mM	0.2776 mL	1.3880 mL	2.7759 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

Methyclothiazide is an orally active antihypertensive agent and a diuretic agent. Methyclothiazide leads to a reduction of the vascular response to the action of endogenous vasoconstricting stimuli, such as Norepinephrine (HY-13715). Methyclothiazide is against voltage-dependent Ca-channel (VDCC) activity in vitro[1][2][3].

IC_{50} & Target

IC50: voltage-dependent Ca-channel (VDCC)

In Vitro

Methyclothiazide (0-100 μM) induces endothelium-dependent inhibition of the vasoconstrictor responses to NE and AVP only in aortas from SHR, and the maximal vasoconstrictive effect of Norepinephrine (HY-13715) and arginine vasopressin (AVP) is decreased by 59% and 32.3 %, respectively[1].

Methyclothiazide (0-100 μM) induces inhibitory effect on the contractile response to Norepinephrine (HY-13715) is abolished by N-nitro-L-arginine (NOLA) but not indomethacin[1].

Methyclothiazide (100 μM) affects the vascular responses to extracellular Ca^{2+} under high-K^+ depolarizing conditions. It can reduce Ca^{2+} contracture in a high-K^+, Ca^{2+}-free solution. The maximal contracture is reduced by 90.4%[1].

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

REFERENCES

[1]. Colas, B., et al., Mechanisms of methyclothiazide-induced inhibition of contractile responses in rat aorta. Eur J Pharmacol, 2000. 408(1): p. 63-7.

[2]. Colas, B., et al., Direct vascular actions of methyclothiazide and indapamide in aorta of spontaneously hypertensive rats. Fundam Clin Pharmacol, 2000. 14(4): p. 363-8.

[3]. Sasaki, S. and R.D. Bunag, Methyclothiazide attenuates salt-induced hypertension without affecting sympathetic responsiveness in Dahl rats. J Cardiovasc Pharmacol, 1983. 5(3): p. 378-83.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA