Trichlormethiazide sodium

Cat. No.: HY-B0235A

CAS No.: 91996-54-2

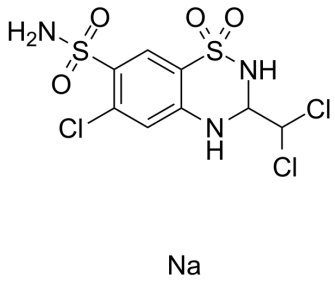
Molecular Formula: $C_8H_8Cl_3N_3NaO_4S_2$

Molecular Weight: 403.65

Target: Others

Pathway: Others

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



BIOLOGICAL ACTIVITY

Description

Trichlormethiazide sodium is an orally active thiazide diuretic, with antihypertensive effect. Trichlormethiazide sodium increases urine volume (UV), Na and K excretion and tends to improve the depressed creatinine clearance (CCRE) in acute renal failure rats model[1][2].

In Vivo

Trichlormethiazide (1 mg/kg; p.o.; once) sodium increases urinary volume, sodium and potassium excretion in rats[1].
Trichlormethiazide (10 mg/kg, i.v.; daily for 5 days) sodium significantly reduces mean arterial pressure (MAP) within 24 h in high salt intake (HS) rats receiving angiotensin II, but does not affect MAP in any other group[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Wistar rats, weighing 170-300 g[1]
Dosage:	1 mg/kg
Administration:	Oral administration, once
Result:	Significantly increased potassium excretion in normal rats. Significantly increased urinary volume, sodium and potassium excretion in cisplatin-induced ARF (acute renal failures) rats.

Animal Model:	Male Sprague-Dawley rats (350-450 g)[2]
Dosage:	10 mg/kg
Administration:	Intravenous injection, daily, for 15 days
Result:	Produced a significant fall in MAP in rats on combined angiotensin II and high salt intake.

REFERENCES

[1]. K Yao, et al. Diuretic effects of KW-3902, a novel adenosine A1-receptor antagonist, in various models of acute renal failure in rats. Jpn J Pharmacol. 1994 Apr;64(4):281-8.

[2]. J R Ballew, et al. Characterization of the antihypertensive effect of a thiazide diuretic in angiotensin II-induced hypertension. J Hypertens. 2001 Sep;19(9):1601-6.

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