Furosemide

Cat. No.: HY-B0135

CAS No.: 54-31-9

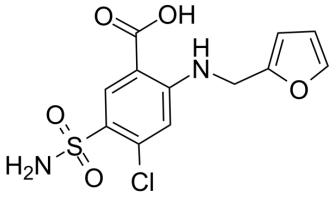
Molecular Formula: $C_{12}H_{11}ClN_2O_5S$

Molecular Weight: 330.74

Target: NKCC; GABA Receptor

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage: 4°C, protect from light
* In solvent : -80°C, 1 year; -20°C, 6 months (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (302.35 mM)
H_2O : < 0.1 mg/mL (insoluble)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.0235 mL	15.1176 mL	30.2352 mL
	5 mM	0.6047 mL	3.0235 mL	6.0470 mL
	10 mM	0.3024 mL	1.5118 mL	3.0235 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 (7.56 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.56 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Furosemide is a potent and orally active inhibitor of $Na^+/K^+/2Cl^-$ (NKCC) cotransporter, NKCC1 and NKCC2[1]. Furosemide is also a $GABA_A$ receptors antagonist and displays 100-fold selectivity for α6-containing receptors than α1-containing receptors. Furosemide acts as a loop diuretic and used for the study of congestive heart failure, hypertension and edema[2].

IC_{50} & Target

IC50: NKCC1 and NKCC2[1]
IC50: $GABA_A$ receptors[2]

In Vitro

Furosemide (500 μM; 72-96 hours) significantly changes the proliferation rates in MKN45 cells (the poorly differentiated human gastric adenocarcinoma cell line). however, it has no effects on MKN28 cells (the moderately differentiated human

	gastric adenocarcinoma cell line). The growth rate of MKN45 cells is larger than that of MKN28 cells[4]. Furosemide (10 μM, 30 μM, 100 μM; 45 min exposure) significantly decreases cation channel activity and [Ca(2+)](i) in human erythrocytes drawn from healthy individuals. Tert-butylhydroperoxide similarly enhances the non-selective cation channels activity, increases [Ca(2+)](i) and triggered cell membrane scrambling, however, the effects is significantly blunted by furosemide again[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Furosemide (intraperitoneal injection; 100 mg/kg; single dose) is injected after kanamycin (KM) (1000 mg/kg) to creat a deaf mouse model in C57BL/6 mouse. After injection, hearing loss and cochlear hair cell damage are evaluated on day 1, day 2 and day 3, respectively. The hearing is markedly deteriorated even from the next day (Day-1 group), OHCs (outer hair cell) morphology of apical, middle and basal turns are disorganized in mice on day3[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Pharmaceut Biomed. 2020, 113870.
- J Orthop Surg Res. 2024 Feb 19;19(1):147.

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REFERENCES

[1]. C M Gillen, et al. Molecular cloning and functional expression of the K-Cl cotransporter from rabbit, rat, and human. A new member of the cation-chloride cotransporter family. J Biol Chem. 1996 Jul 5;271(27):16237-44.

[2]. S A Thompson, et al. Residues in transmembrane domains I and II determine gamma-aminobutyric acid type AA receptor subtype-selective antagonism by Furosemide sodium. Mol Pharmacol. 1999 Jun;55(6):993-9.

[3]. Shin Hye Kim, et al. Novel Peptide Vaccine GV1001 Rescues Hearing in Kanamycin/Furosemide sodium-Treated Mice. Front Cell Neurosci. 2018 Jan 19;12:3.

[4]. Atsushi Shiozaki , et al. Furosemide sodium, a blocker of Na+/K+/2Cl- cotransporter, diminishes proliferation of poorly differentiated human gastric cancer cells by affecting G0/G1 state. J Physiol Sci. 2006 Dec;56(6):401-6.

[5]. Yuliya V Kucherenko, et al.Inhibitory effect of Furosemide sodium on non-selective voltage-independent cation channels in human erythrocytes.Cell Physiol Biochem. 2012;30(4):863-75.

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