Lidocaine

Cat. No.: HY-B0185

CAS No.: 137-58-6

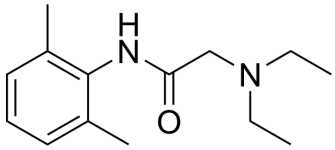
Molecular Formula: $C_{14}H_{22}N_2O$

Molecular Weight: 234.34

Target: Sodium Channel; MEK; ERK; NF-κB; Apoptosis

Pathway: Membrane Transporter/Ion Channel; MAPK/ERK Pathway; Stem Cell/Wnt; NF-κB; Apoptosis

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 (426.73 mM)
H_2O : ≥ 5 mg/mL (21.34 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	4.2673 mL	21.3365 mL	42.6730 mL
	5 mM	0.8535 mL	4.2673 mL	8.5346 mL
	10 mM	0.4267 mL	2.1337 mL	4.2673 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 4.35 mg/mL (18.56 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (10.67 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (10.67 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.67 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Lidocaine (Lignocaine) inhibits sodium channels involving complex voltage and using dependence[1]. Lidocaine decreases growth, migration and invasion of gastric carcinoma cells via up-regulating miR-145 expression and further inactivation of MEK/ERK and NF-κB signaling pathways. Lidocaine is an amide derivative and has potential for the research of ventricular arrhythmia[2].

IC_{50} & Target	MEK	ERK	NF-κB

In Vitro

Lidocaine (Lignocaine) (10 nM; 48 hours) decreases significantly cell proliferation[2].
?Lidocaine (1-10 nM; 24-72 hours) inhibits cell viability and achieves the most suppressing effects at the concentration of 10?nM and treatment time 48?hours[2].
?Lidocaine (10 nM; 48 hours) increases significantly the apoptotic cell rate[2].
?Lidocaine (10 nM; 48 hours) down-regulates Cyclin D1 and up-regulates p21 expression significantly[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[2]

Cell Line:	The human gastric cancer cell line MKN45
Concentration:	10 nM
Incubation Time:	48 hours
Result:	Decreased significantly cell proliferation.

Cell Viability Assay[2]

Cell Line:	The human gastric cancer cell line MKN45
Concentration:	1, 5 and 10 nM
Incubation Time:	24, 48, 72 hours
Result:	Inhibited MKN45 cell viability.

Apoptosis Analysis[2]

Cell Line:	The human gastric cancer cell line MKN45
Concentration:	10 nM
Incubation Time:	48 hours
Result:	Increased significantly the apoptotic cell rate.

Western Blot Analysis[2]

Cell Line:	The human gastric cancer cell line MKN45
Concentration:	10 nM
Incubation Time:	48 hours
Result:	Down-regulated Cyclin D1 and up-regulated p21 expression significantly.

In Vivo

Lidocaine (Lignocaine) causes completely reversible tail nerve block in rats. Mechanical nociception block produced by lidocaine has slower onset and faster recovery compared with thermal nociception block[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Methods. 2021 Jul;18(7):788-798.

- J Neuroinflammation. 2017 Nov 2;14(1):211.
- Stem Cell Res Ther. 2021 Feb 4;12(1):107.
- PLoS Pathog. 2023 Feb 3;19(2):e1011126.
- Int Immunopharmacol. 2023 Jan 11;115:109706.

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REFERENCES

[1]. Cummins TR, et al. Setting up for the block: the mechanism underlying lidocaine's use-dependent inhibition of sodium channels. J Physiol. 2007 Jul 1;582(Pt 1):11.

[2]. Sui H, et al. Lidocaine inhibits growth, migration and invasion of gastric carcinoma cells by up-regulation of miR-145. BMC Cancer. 2019 Mar 15;19(1):233.

[3]. Li Z, et al. Evaluation of the antinociceptive effects of lidocaine and bupivacaine on the tail nerves of healthy rats. Basic Clin Pharmacol Toxicol. 2013 Jul;113(1):31-6.

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