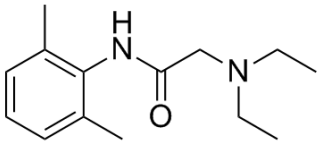
Lidocaine

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
Pathway:	Membrane Transporter/Ion Channel; MAPK/ERK Pathway; Stem Cell/Wnt; NF-κB
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (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

Solvent / Mass / 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: **10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline**
   Solubility: ≥ 2.5 mg/mL (10.67 mM); Clear solution
2. 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
3. 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 a commonly used local anesthetics of amide derivative, a drug to treat ventricular arrhythmia and an effective tumor-inhibitor[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].

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].

CUSTOMER VALIDATION

- **J Neuroinflammation**. 2017 Nov 2;14(1):211.
- **J Phys D Appl Phys**. 2019 Aug.

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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