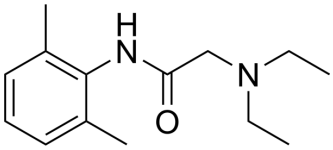
Lidocaine (GMP)

Cat. No.:	HY-B0185G
CAS No.:	137-58-6
Molecular Formula:	$C_{14}H_{22}N_2O$
Molecular Weight:	234.34
Target:	Apoptosis; Sodium Channel; MEK; ERK; NF-κB
Pathway:	Apoptosis; Membrane Transporter/Ion Channel; MAPK/ERK Pathway; Stem Cell/Wnt; NF-κB
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Lidocaine (GMP) is Lidocaine (HY-B0185) produced by using GMP guidelines. GMP small molecules work appropriately as an auxiliary reagent for cell therapy manufacture. Lidocaine 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].
In Vitro	Lidocaine GMP (Lignocaine) (10 nM; 48 hours) decreases significantly cell proliferation[2]. Lidocaine GMP (1-10 nM; 24-72 h) inhibits cell viability and achieves the most suppressing effects at the concentration of 10 nM and treatment time 48 hours[2]. Lidocaine GMP (10 nM; 48 h) increases significantly the apoptotic cell rate[2]. Lidocaine GMP (10 nM; 48 h) down-regulates Cyclin D1 and up-regulates p21 expression significantly[2]. Lidocaine (100 μM, 200 μM, 28 d) slowed down the conduction velocity (CV) in hPSC lines[4]. Lidocaine (0.1-2000 μM, 5 min) exhibits limited use-dependent block (UDB) in hiPSC-derived cardiomyocytes[5]. Lidocaine (30 μM) reduces QT interval of LQTS-CMs to a normal level[6]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Lidocaine GMP (Lignocaine) causes completely reversible tail nerve block in rats. Mechanical nociception block produced by Lidocaine GMP 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.

[4]. Kadota S, et al. Development of a reentrant arrhythmia model in human pluripotent stem cell-derived cardiac cell sheets. Eur Heart J. 2013 Apr;34(15):1147-56.

[5]. Potet F, et al. GS-967 and Eleclazine Block Sodium Channels in Human Induced Pluripotent Stem Cell-Derived Cardiomyocytes. Mol Pharmacol. 2020 Nov;98(5):540-547.

[6]. Wang F, et al. In Vitro Drug Screening Using iPSC-Derived Cardiomyocytes of a Long QT-Syndrome Patient Carrying KCNQ1 & TRPM4 Dual Mutation: An Experimental Personalized Treatment. Cells. 2022 Aug 11;11(16):2495.

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