Lidocaine (603): sc-57901



The Power to Question

BACKGROUND

Lidocaine is a common amino amide-type local anesthetic as well as a Class Ib antiarrhythmic agent. Lidocaine has a longer duration and a more rapid onset of action than amino ester-type local anesthetics. It functions by blocking fast sodium channels in the cell membrane, thereby effectively altering depolarization in neurons. With enough Lidocaine, the membrane will not transmit an action potential, leading to its anesthetic effects. When Lidocaine blocks a cardiac action potential, it decreases automaticity by reducing the slope of phase 0 of depolarization with little effect on the PR interval, QRS complex or QT interval. Systemic exposure to large amounts of Lidocaine may result in negative central nervous system and cardiovascular effects. CYP1A2, a liver enzyme, metabolizes about 90 percent of Lidocaine into the pharmacologically-active metabolites monoethylglycinexylidide and glycinexylidide. Lidocaine has a molecular weight of 234.34 g/mol and a half life of 1.5 to 2 hours.

REFERENCES

- Whitcomb, D.C., Gilliam, F.R., Starmer, C.F. and Grant, A.O. 1989. Marked QRS complex abnormalities and sodium channel blockade by propoxyphene reversed with Lidocaine. J. Clin. Invest. 84: 1629-1636.
- Murasato, Y., Nagamoto, Y., Urabe, T., Kuraoka, F., Nakashima, Y. and Kuroiwa, A. 1997. Effects of Lidocaine and diltiazem on recovery of electrophysiologic activity during partial reperfusion following severe myocardial ischemia in canine hearts. J. Electrocardiol. 30: 113-125.
- Wang, J.S., Backman, J.T., Wen, X., Taavitsainen, P., Neuvonen, P.J. and Kivistö, K.T. 2000. Fluvoxamine is a more potent inhibitor of Lidocaine metabolism than ketoconazole and erythromycin *in vitro*. Pharmacol. Toxicol. 85: 201-205.
- 4. Sarraf, G., Barrett, T.D. and Walker, M.J. 2003. Tedisamil and Lidocaine enhance each other's antiarrhythmic activity against ischaemia-induced arrhythmias in rats. Br. J. Pharmacol. 139: 1389-1398.
- 5. Li, L., Nikolski, V. and Efimov, I.R. 2004. Effects of Lidocaine on shock-induced vulnerability. J. Cardiovasc. Electrophysiol. 14: S237-248.
- Sandtner, W., Szendroedi, J., Zarrabi, T., Zebedin, E., Hilber, K., Glaaser, I., Fozzard, H.A., Dudley, S.C. and Todt, H. 2004. Lidocaine: a foot in the door of the inner vestibule prevents ultra-slow inactivation of a voltage-gated sodium channel. Mol. Pharmacol. 66: 648-657.
- 7. Haga, H.A., Lykkjen, S., Revold, T. and Ranheim, B. 2006. Effect of intratesticular injection of Lidocaine on cardiovascular responses to castration in isoflurane-anesthetized stallions. Am. J. Vet. Res. 67: 403-408.
- 8. Saghaei, M., Reisinejad, A. and Soltani, H. 2006. Prophylactic versus therapeutic administration of intravenous Lidocaine for suppression of post-extubation cough following cataract surgery: a randomized double blind placebo controlled clinical trial. Acta Anaesthesiol. Taiwan 43: 205-209.
- 9. Newton, D.J., McLeod, G.A., Khan, F. and Belch, J.J. 2007. Mechanisms influencing the vasoactive effects of Lidocaine in human skin. Anaesthesia 62: 146-150.

SOURCE

Lidocaine (603) is a mouse monoclonal antibody raised against Lidocaine.

PRODUCT

Each vial contains 100 μ l ascites containing lgG_1 with < 0.1% sodium azide.

APPLICATIONS

Lidocaine (603) is recommended for detection of Lidocaine by solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

STORAGE

For immediate and continuous use, store at 4° C for up to one month. For sporadic use, freeze in working aliquots in order to avoid repeated freeze/thaw cycles. If turbidity is evident upon prolonged storage, clarify solution by centrifugation.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.

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