

Abstract

The Role of Lidocaine in the Dunning Model Rat Prostate Cancer Cells: Cell Kinetics and Motility [†]

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[†] Presented at the 2nd International Conference on Natural Products for Cancer Prevention and Therapy, Kayseri, Turkey, 8–11 November 2017.

Published: 15 November 2017

Abstract: Deaths resulting from prostate cancer are due to metastases rather than primary tumor. Therefore, understanding metastatic mechanisms play a significant role. It has been determined that metastatic progression can be avoided by the voltage-gated sodium channels (VGSCs) in cell membranes. We aimed to evaluate the effects of Lidocaine, a VGSC blocker, on the cell proliferation of rat prostate cancer cells (Mat-LyLu and AT-2) and the lateral motility, which indicates metastases. The effect of lidocaine at different concentrations (0.1–10 mM) on the proliferation of cells was determined by the MTT method, and the effects on DNA synthesis was determined by the autoradiography. Motility of cells was realized with the wound heal method, and tetrodotoxin was used as the positive control. We have obtained, lidocaine decreased the proliferations of high metastatic Mat-LyLu cells and low metastatic AT-2 cells from 0.5 mM and 1 mM, respectively. Labeling index results are also parallel with proliferation results. While lidocaine decreased the motility of Mat-LyLu cells expressing Nav.1.7 VGSC ($p < 0.05$), it did not cause a change in the motility of AT-2 cells. It has been demonstrated for the first time that lidocaine has the potential to be used as an ion channel blocker in rat prostate cancer metastases.

Keywords: Lidocaine; Prostate cancer; Mat-LyLu cells; AT-2 cells; Lateral Motility; Cell proliferation; Labelling index



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