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ST6GAlI Regulating the Chemosensitivity Does Not Directly Relate to the Accumulation of Doxorubicin of Breast Cancer *In Vitro*

Lili JIA [1], Xiuzhen HE [1], Zhenzhen LIAN [1], Xiaoyu WANG [2], Naihua LIU [1], Yongbo YAN [1], Hua LI [1], Kayiu WAN [1] & Shaoqiang LIN *[1]

[1] *Wenzhou Medical College, Wenzhou City, Zhejiang Province 325035, China.*
[2] *The First Affiliated Hospital of Jinan University, Guangzhou City, Guangdong Province 510000, China.*

***SUMMARY*. In tumor treatment, low bioavailability and high reverse effect remains a formidable challenge. It is urgently needed to develop novel and effective therapies. We intend to develop cationic liposomes as a novel drug delivery system. Flow Cytometer was used to detect the doxorubicin accumulation of MDA-MB-435 cells, sh-ST6Gal I cells and HUVECs. Then we further detected the apoptosis induced by doxorubicin, cationic liposomes and anionic liposomes. The cationic liposomes with positive zeta potential have affinity to tumor cells by electrostatic interaction, increase the cell uptake of doxorubicin and cell apoptosis. Downregulation of ST6Gal I significantly enhances the sensitivity of anticancer drug to breast cancer and increased the apoptosis percentage. Combined used inhibition of ST6GalI expression and chemotherapy may be a better strategy for some tumors with overexpressed of ST6GalI.**

KEYWORDS: Cationic liposomes, Cell surface charge,doxorubicin, ST6Gal I.

* Author to whom correspondence should be addressed. *E-mail* address: shaotsiang@163.com

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