

عنوان مقاله:

Investigating the effect of microRNA-6895 in resistance of breast cancer cells to doxorubicin chemotherapy:
Bioinformatics analysis

محل انتشار:

سومین کنگره بین المللی و پانزدهمین کنگره ملی ژنتیک ایران (سال: 1397)

تعداد صفحات اصل مقاله: 5

نویسندگان:

Samira Rahimi Rad - *Medical Genetic Department, National Institute of Genetic Engineering and Biotechnology, Tehran, Iran*

.Shima Rahimi Rad - *Genetic Department, Faculty of Science, University of Shahrekord, Shahrekord, Iran*

Mohammad Navaderi - *Medical Genetic Department, National Institute of Genetic Engineering and Biotechnology, Tehran, Iran*

.Aysan Jafari Harandi - *Nourdanesh Institute of Higher Education, Iran*

Sharareh khorami ruz - *Microbiologist in American Hospital Dubai_oud metha-po BOX5566*

خلاصه مقاله:

Resistance of breast cancer cells to chemotherapy is a major barrier of successful treatment. Evidence have indicated the impact roles of gene function mediated by short noncoding RNA in chemotherapy resistance. The MDR1 gene also known as P-Glycoprotein is a member of the superfamily of ATP-binding cassette (ABC) transporters. This gene is responsible for decreased drug accumulation in multidrug-resistant cells by affecting the susceptibility to Doxorubicin (DOX). The present study, we investigated the MDR1 gene expression modulators which affects the resistance of the cancer cells to chemotherapeutic drug doxorubicin. Method Using CTD database (<http://ctdbase.org/>) the interaction of MDR1 gene with DOX were confirmed. To identify the miRNA based regulators of MDR1 gene we constructed the miRNA-mRNA interaction network using Cytoscape version 3.4. Next, DIANA tools microRNA target databases (<http://diana.imis.athena-innovation.gr/DianaTools/index.php>) and single nucleotide polymorphism (SNP) searching tools (<https://www.ncbi.nlm.nih.gov/projects/SNP/>) were used for studying gene expression regulation by the effect of SNP in miRNA seed region associated with MDR1 gene. Result Our microRNA target analysis of MDR1 gene showed that has-miR-6895-5p regulates this gene expression (Fig.1). In a complementary analysis, we identified deletion of bases -/ACAGAGAG (rs56794014) located within miR-6895-5p target site in 3' UTR of MDR1 gene (Fig.2). This variant may result in a blockage of miRNA – Based downregulation of MDR1 gene which is results in the increased chemoresistance property of cancer cells to DOX. Discussion Our results indicating that MDR1 gene deletion in miR-6895-5p target site may have significant implications for identifying the therapeutic strategies aiming to overcome breast cancer cell resistance.

کلمات کلیدی:

.Cancer Cell, Drug Resistance, Bioinformatics, microRNA

