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PII: S0344-0338(17)31174-3
DOI: https://doi.org/10.1016/j.prp.2018.02.017
Reference: PRP 52007

To appear in:

Received date: 19-11-2017
Revised date: 2-2-2018
Accepted date: 18-2-2018

Please cite this article as: Tian-tian Li, Xiang Gao, Li Gao, Bin-liang Gan, Zu-cheng Xie, Jing-jing Zeng, Gang Chen, Role of upregulated miR-136-5p in lung adenocarcinoma: a study of 1242 samples utilizing bioinformatics analysis, Pathology - Research and Practice https://doi.org/10.1016/j.prp.2018.02.017

Role of upregulated miR-136-5p in lung adenocarcinoma: a study of 1242 samples utilizing bioinformatics analysis

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Abstract:

Background: It is generally acknowledged that miRNAs play pivotal roles in the initiation and development of cancer. The aim of the current study is to investigate the clinicopathological role of miR-136-5p in lung adenocarcinoma and its underlying molecular mechanism. **Materials and methods:** Data of a cohort of 1242 samples were provided by the Gene Expression Omnibus and The Cancer Genome Atlas to evaluate miR-136-5p expression in lung adenocarcinoma. A comprehensive meta-analysis integrating the expression data from all sources was performed, followed by a summary receiver operating curve plotted to appraise the upregulated expression of miR-136-5p in lung adenocarcinoma. Candidate targets of miR-136-5p were launched by the intersection of differentially expressed genes in The Cancer Genome Atlas and genes predicted by 12 web-based platforms. Then, hub genes were illustrated by a protein-protein interaction network. Furthermore, Kyoto Encyclopedia of Genes and Genomes, Gene Ontology and Protein Analysis Through Evolutionary Relationships analyses of potential target genes were carried out via bioinformatics tools. **Results:** MiR-136-5p expression was upregulated in lung adenocarcinoma versus normal tissues (standard mean difference=0.43, 95% confidence interval: 0.27-0.58). The summary receiver operating characteristic curve further verified the upregulation of miR-136-5p in lung adenocarcinoma (area under curve=0.7459). A total of 311 candidate target genes of miR-136-5p were gathered to create a protein-protein interaction network. Molecular mechanism analysis unveiled the potential miR-136-5p target genes participated in cell adhesion molecules, focal adhesion, complement and coagulation cascades and blood coagulation. **Conclusion:** MiR-136-5p is overexpressed in lung adenocarcinoma and is involved in the molecular mechanism of lung adenocarcinoma via suppressing the expressions of downstream targets, especially claudin-18, sialophorin and syndecan 2 that participate in cell adhesion.

Key words: lung adenocarcinoma; miR-136a-5p; meta-analysis; bioinformatics; cell adhesion molecules.