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摘要:

目的: 探讨hsa miR 132 (miR 132) 对人食管癌细胞增殖和侵袭的影响及其可能的机制。方法: 构建pCDNA3.1(+) miR 132表达载体并转染高转移潜能的人食管癌细胞KYSE150, G418筛选稳定过表达miR 132的KYSE150细胞。琼脂克隆形成实验和Transwell小室实验检测KYSE150细胞的增殖和侵袭, Real time PCR和Western blotting检测miR 132和叉头框蛋白A1基因 (forkhead box proteins A1, FOXA1) 在KYSE150细胞中的表达, 双荧光素酶报告基因分析和Western blotting检测过表达miR 132对 FOXA1 的调控作用。结果: 成功构建pCDNA3.1(+) miR 132质粒, 获得稳定过表达miR 132的KYSE150细胞。亲本KYSE150细胞中miR 132低表达, 而FOXA1蛋白高表达。与转染空载体和未转染KYSE150细胞相比, 转染pCDNA3.1(+) miR 132不影响KYSE150细胞的增殖 (13.9±0.33 vs 15.4±0.11、17.1±0.20, P >0.05), 但细胞体积较小且表面比较光滑; 其可显著降低KYSE150细胞的侵袭能力 [(55±1.6) vs (129.0±3.1)、(124.0±2.8)个细胞, P <0.01]。miR 132能够作用于 FOXA1 3' UTR, 从而抑制内源性 FOXA1 的表达。结论: 食管癌KYSE150细胞低表达miR 132, 外源性过表达miR 132可负向调控 FOXA1 的表达, 从而抑制KYSE150细胞的侵袭能力。

关键词: [食管癌](#) [侵袭](#) [增殖](#) [has-miR-132](#) [FOXA1基因](#)

Hsa-miR-132 affects proliferation and invasion of esophageal carcinoma cells by targeting FOXA1 gene [Download Fulltext](#)

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Abstract:

Objective: To investigate the effect of hsa miR 132 (miR 132) on proliferation and invasion of human esophageal carcinoma cells, and further explore its possible mechanism. Methods: pCDNA3.1(+) miR 132 plasmid was constructed, and KYSE150 cells stably overexpressing miR 132 were selected through G418 screening. Soft agar colony formation test and Transwell assay were performed to analyze the proliferation and invasion of KYSE150 cells. Expression of miR 132 and forkhead box proteins A1 (FOXA1) in KYSE150 cells were examined by real time PCR and Western blotting. The regulatory effect of miR 132 overexpression on FOXA1 was further studied by reporter gene assay and Western blotting. Results: pCDNA3.1(+) miR 132 plasmid was successfully constructed, and KYSE150 cells stably overexpressing miR 132 was established. In parental KYSE150 cells, miR 132 expression was low, while FOXA1 expression was high. Compared to mock transfected KYSE150 cells and untransfected KYSE150 cells, pCDNA3.1(+) miR 132 transfection did not affect the proliferation of KYSE150 cells (13.9±0.33 vs 15.4±0.11, 17.1±0.20, P >0.05), however tumor size reduced and tumor appeared smoother after pCDNA3.1(+) miR 132 transfection. Furthermore, overexpression of miR 132 significantly suppressed the invasion of KYSE150 cells (55±1.6 vs 129.0±3.1, 124.0±2.8, P <0.01), and miR 132 decreased the expression of endogenous FOXA1 by targeting FOXA1 3' UTR. Conclusion: Esophageal carcinoma KYSE150 cells express low level of miR 132, and overexpression of miR 132 can negatively regulate the expression of FOXA1 and suppressed the invasion of KYSE150 cells.

Keywords: [esophageal carcinoma](#) [invasion](#) [proliferation](#) [hsa-miR-132](#) [FOXA1](#)

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