**58. miR-206 Targets MALAT1 to Suppress Cell Progression of Ectopic Endometrial Stromal Cells in Endometriosis**

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Abstract

Background: miR-206 was reported to be a tumor suppressor in bladder cancer. In this study, we explore the expression and function of miR-206 in endometriosis (EM).

Methods: 40 EM patients undergoing total hysterectomy were selected as the experimental group. RT-qPCR assay was adopted to detect the expression of MALAT1 and miR-206 in EM. Cell proliferation was detected by EdU incorporation and colony formation assay. Cell migration and invasion viability of ESCs were examined by transwell assay and wound healing assay. Flow cytometry was carried out to assess cell apoptosis of ESCs. The protein expressions of Bcl-2 and Bax were examined by western blot assay. The relationship between miR-206 and MALAT1 was verified by the dual-luciferase reporter assay and RNA pull-down assay.

Results: In this work, miR-206 was found to be downregulated in EM. Functional experiments displayed that miR-206 mimic repressed cell proliferation, migration, and invasion of ESCs and promoted cell apoptosis of ESCs. Furthermore, miR-206 mimic reduced the expression of Bcl-2 but enhanced the expression of Bax. MALAT1 was found to be upregulated in EM. Furthermore, MALAT1 was indicated to be a target of miR-206. Additionally, MALAT1 was found to alleviate the influence of miR-206 on cell progression of ESCs. Furthermore, miR-206 inhibited tumor growth *in vivo*.

Conclusion: This study indicated that miR-206 inhibited cell progression by regulating MALAT1 in EM. Hence, miR-206 was suggested to be a possible target for EM treatment.

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